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CANCER OF THE OVARY*

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BOSTON

IN the *Southern Medical Journal* for February, 1937, there is an article[1] on cancer of the ovary, with a review of all the cases treated at the Massachusetts General Hospital from 1901 through 1931, a period of thirty-one years. Because the results of treatment of these malignant epithelial tumors of the ovary were so poor the series was brought up to 1934, a period of thirty-three years. The outlook for patients with this disease is so hopeless that it seemed pertinent to present the subject here in the hope that a greater interest could be stimulated in early diagnosis and in earlier and more radical treatment. Cancer of the cervix shows absolute curability of 24 per cent in the series at the Massachusetts General Hospital, and cancer of the breast with axillary involvement, a 25 per cent arrest for five years. In the recent Pondville series[2] the five-year survivals for cervical cancers have been raised to 34.5 per cent through a combined use of x-ray and radium. The five-year end results for malignant epithelial lesions of the ovary are worse than the above series, and show only 24 patients out of 147, or 16 per cent, living without demonstrable disease. Thus the seriousness of this group of tumors is easily appreciated. Yet if earlier symptoms could be interpreted and earlier diagnoses be made, the end results should be much better, for the tumors in their early stages are easily dealt with.

MATERIAL

During the years 1901–1933 inclusive there were 147 patients with cancer of the ovary. The history of each case was carefully abstracted, the slides of each tumor were reviewed and studied, and any doubtful tumors were taken for review by the hospital pathologist, Dr. Tracy B. Mallory. If a tumor was classified as malignant on the hospital record but if this diagnosis was not confirmed on the review of the slides, it was discarded. Furthermore, some patients with cystadenomas of the ovary which were believed to be benign were included in the malignant group after restudy of microscopic sections. No case was accepted for this series if the diagnosis was made on a biopsy specimen from the peritoneum unless the surgeon was certain that the lesion originated in the ovary; and no metastatic tumor of known or unknown origin was accepted. Thus all cancers of the ovary with the primary lesion in the endometrium were discarded, as well as tumors metastasizing from the stomach (Krukenberg) or intestine. There were a few patients with generalized peritoneal implantations which were called malignant in the old records; if these lesions looked benign microscopically and the patient was still living after five years, the diagnosis was regarded as incorrect and a proper one of benign papillary cystadenoma of the ovary with peritoneal implantation was substituted. All tumors of peculiar or "odd" types were excluded, such as granulosal-cell carcinomas, dysgerminomas, Brenner tumors, sarcomas, teratomas (dermoids), and so forth. If no slides were available for study, the case was excluded at once. In all, more than 250 tumors of malignant types were reviewed and all but 147 were discarded. The remaining group is a selected one, but includes as few benign and "odd" cases as is possible. The history and end result, with microscopic study of the tumor by the hospital pathologist and surgeon, should be made the ideal for reports of the end results of cancer treatment. The possibility of misinterpretation in the pathological laboratory when clinical information is not available is very great. Much more valuable information would be available from the literature if no reports were presented without a complete recheck of each case at the time the reports are made.

EMBRYOLOGY OF THE OVARY

Because of the multiplicity of tumors that originate in the ovary, an abbreviated description of its embryology is presented.

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The ovary arises as a mass of mesenchyme on the back wall of the abdominal cavity. Germinal epithelium from the pelvic or celomic epithelium surrounds this mass. The ova arise either from the hind gut or the germinal epithelium and migrate into the mass. The connective tissue surrounding the ova becomes highly specialized and forms the granulosal and thecal cells of the follicles. Thus in the depth of the gonad and from mesenchyme arise structures that can eventually become epithelial — for instance the lining of certain follicle cysts. It is possible that the primitive ova are not the ones that develop into mature ova with their surrounding follicular apparatus. It may be that new eggs arise in the germinal epithelium during adult life, find their way into the cortex of the ovary and develop there.

The gonad is probably neither ovarian nor testicular at the beginning, but its development depends on the presence of sperm or ova. If abnormal ova are present, cells that ordinarily become granulosal or thecal may become testicular or tubular (Sertoli). The gonad has also been considered as testicular at first and later cupped by ovarian tissue; the testicular tissue then atrophies and the organ becomes an ovary. Either possibility explains the presence of testicular tissue in the ovary. Thus it becomes a source of tumors of the male type.

The adrenals and the kidneys develop in close proximity to the gonad, and the possibility of the inclusion of a few cells of these organs is great. Certain peculiar tumors of the ovary suggest a source such as this. The special tumors that we recognize now point to such possible sources — granulosal cell, thecal cell, testicular, tubular, Leydig cell, and so forth. All tumors of special types are excluded from this group for the more highly specialized the tumor the less is its malignancy. In the group accepted there must be many tumors that will be defined as special tumors as our knowledge increases. Therefore it may be that many of our surviving patients were blessed with highly specialized but at present unrecognized tumors. The exclusion of such lesions would greatly alter the results.

SYMPTOMS AND DIAGNOSIS

Ovarian cancer often gives no symptoms until it is well advanced. Frequently increase in size of the abdomen due to tumor is considered as increasing weight and heaviness due to the age of the patient. There may be early a sense of vaginal discomfort and occasionally a slight gastrointestinal upset, but later the chief symptoms are pelvic pain and discomfort, loss of weight and urinary difficulty. Ascites is not uncommon, but it must be remembered that a solid pelvic tumor with ascites and even hydrothorax does not indicate a hopeless prognosis, because fibromas of the ovary, benign cysts and even fibroids may be accompanied by ascites and fluid in the chest.

Age. About half of all patients with ovarian tumors are in the age group of thirty to fifty, more in the older decade than in the younger. Ten per cent are from ten to thirty, while slightly over 40 per cent are from fifty to seventy. In the most malignant group 51 per cent fall into the thirty to fifty group, while 45 per cent were found to be between fifty and seventy. Ovarian cancer is a tumor of older women.

Marital History. In this series 119 patients, or 81 per cent, were married; of the married women but 63 per cent had had children. This is a low incidence of fertility, as the usual figure is approximately 90 per cent. In nearly all statistical studies this finding is persistent. Lynch's[3] paper, written in 1936, showed that but 69 per cent of his patients had had children. These figures in a nearly comparable series of cases tend to show that nonfertility is an important factor. Therefore it is necessary to consider the possibility of some sort of congenital defect of the ovary in this group of patients, for if a congenital developmental defect were present there should be an increased chance for the growth of left-over cells and, hence, for the production of abnormal growths.

Menstrual History. In this series of 147 patients, 48, or 33 per cent, of those still menstruating had regular menstrual periods. Eight, or 5 per cent, were irregular; 18 or 12 per cent, did not give this information in their hospital histories. Seventy-three, or 50 per cent, had had the menopause. Of those past the menopause 15, or 10 per cent, had bleeding as a symptom. Probably the ovary was still secreting enough hormone to affect the endometrium so that the bleeding occurred for physiological reasons. It is doubtful that "congestion" was responsible. It is conceivable that some of the peritoneal implants which disappear after the main tumor is removed do so because ovarian hormone stimulation has been stopped. Many reports have been made of the hormone content of ovarian cyst fluids, and certainly in many the amount of estrin present might account for endometrial changes. Abnormal bleeding after the menopause in the presence of a pelvic mass should immediately make one consider the possibility of ovarian cancer.

TYPES OF TUMORS: GROSS AND HISTOLOGIC

The group of tumors were of all sorts — large, small, papillary, smooth-walled, cystic, solid, solid

and cystic, white, yellow, hemorrhagic, and so forth. There is no way of predicting what the histological picture will be from the gross appearance of the tumor, except that the so-called pseudomucinous tumor has typical pseudomucinous epithelium; whether or not it is malignant is impossible to say. Histologically three types of epithelium were recognized—the pseudomucinous, the endometrial and a large unclassified group. The easiest type to differentiate histologically is the pseudomucinous, with its high columnar cells containing large amounts of pseudomucin. The solid pseudomucinous tumor is usually made up of masses of abnormal but yet recognizable pseudomucinous epithelium. This solid type of tumor is a rarity. Another type that grows in papillary and solid forms resembles the epithelium of the endometrium. Inasmuch as the celomic epithelium produces the covering of the ovary and the Müllerian ducts, and since these ducts give rise to the endometrium, it is not surprising to find an endometrium-like tumor. Some areas in such a tumor may not suggest endometrium, and this finding probably indicates the extremely atypical character of its growth. Tumors of the so-called unclassified type are histologically different and complex, though after long study certain types are becoming more and more familiar. Their epithelium varies from well-differentiated to very undifferentiated cell types. The most bizarre and peculiar types of cells and structure may be present. This group is one of exclusion, for if the tumor could not be classified as pseudomucinous or endometrial it was placed in the unclassified group.

The criteria of malignancy used were the evidence of atypicality of cells, the presence of mitotic figures, the invasion of the walls of cysts with growing cells, undifferentiation and, in the papillary growths, the presence of areas which if seen alone would be classified as adenocarcinoma. There was no doubt of the malignancy of the tumors included in the group under study. The end results as presented seem to justify the grouping. No doubt a few mistakes have been made, but the malignancy or benignity of a tumor was usually decided from the history and the microscopic slide, and the end result was then checked. If there was a doubt as to malignancy and if the patient was living and well without disease, this tumor was classed as benign.

Grossly the tumors were divided into the following three groups: solid, and solid and cystic; malignant papillary cystadenoma with areas of adenocarcinoma; and malignant papillary cystadenoma. These divisions are primarily clinical and the separation of the two types of malignant papillary cystadenomas usually necessitates histological differentiation. Combining the latter two, however, makes two fairly easily separated groups. The difference in prognosis between the solid group and the cystadenoma group suggests that two groups are sufficient. The division of the cystic groups is presented because of their histological difference.

SOLID, AND SOLID AND CYSTIC, CARCINOMA OF THE OVARY

This group is by far the most serious. Its operability is small and the prognosis very poor. By the solid type is meant a tumor with no cysts except those caused by areas of necrosis. The solid and cystic tumors are those with thick walls invaded by cancer and with large masses of cancer tissue present inside or outside the cyst. There may be marked necrosis and liquefaction or a huge growth of carcinoma from and into the wall of a malignant cyst. The tumor may be large or small, but must have at least half the bulk made up of solid cancer tissue. These tumors grow as medullary carcinoma, adenocarcinoma or papillary adenocarcinoma. The type of cell may be any one of the three types described above. Certain cases in this group may be the result of a malignant papillary cystadenoma which has grown beyond the cyst stage into the solid stage. The preoperative diagnosis is difficult. The symptoms and physical findings are presented in Table 1. A study of the cases makes one feel that any woman past the age of forty who has in either vault a hard area suggestive of an ovarian lesion must be urged to be operated on at once. There is no method of making an accurate diagnosis except by operation or perhaps by peritoneoscopy. These tumors may be accompanied by abnormal bleeding before or after the menopause. In this series 37 per cent were bilateral, and it is probable that more were or had been bilateral. It is certain that in patients with solid cancer of the ovary it is

TABLE 1. *Summary of Clinical Data on 67 Cases with Solid Carcinomas of the Ovary.*

CLINICAL DATA	PERCENTAGE OF CASES POSITIVE
Well developed and nourished	58
Pain	67
Abdominal swelling	49
Loss of weight	40
Ascites	36
Urinary symptoms	27
Duration 1 year or less before operation	64
Age 30 to 50	51
Fertility (married women)	64
Catamenia:	
Regular	33
Menopause passed	51
Abnormal bleeding	30
After menopause	7

necessary that both ovaries, the uterus and cervix be removed if possible. No attempt should be made to conserve ovarian tissue. These tumors metastasize far and wide, and patients may return after the five-year period from operation has been passed with metastatic nodules in various parts of their bodies. X-ray treatment is of some value in prolonging the lives of patients in this group, but a cure cannot be expected.

MALIGNANT PAPILLARY CYSTADENOMA WITH AREAS OF ADENOCARCINOMA

The division of malignant papillary cysts into two groups has justification, for the end results show that the type with areas of adenocarcinoma is more serious than a simple malignant papillary cystadenoma. Any papillary cystic tumor that microscopically has an area that suggests adenocarcinoma in its walls is included in this group. The pseudomucinous cysts are the least malignant and the endometrial type the most. The lesion spreads, as do the solid types, but not quite so widely and rapidly. The same type of radical surgery should be used for this tumor.

TABLE 2. *Summary of Clinical Data on 80 Cases with Malignant Papillary Cystadenomas, with and without Areas of Adenocarcinoma.*

CLINICAL DATA	PERCENTAGE OF CASES POSITIVE
Well developed and nourished	53
Pain	55
Abdominal swelling	58
Loss of weight	54
Ascites	50
Urinary symptoms	44
Duration 1 year or less before operation	56
Age 30 to 50	48
Fertility (married women)	63
Catamenia:	
Regular	33
Menopause passed	48
Abnormal bleeding	20
After menopause	13

MALIGNANT PAPILLARY CYSTADENOMA

This tumor is not so malignant as the other two; yet its treatment is not satisfactory. Here again the pseudomucinous type is the least malignant and the endometrial the most. Metastasis is not so common as it is in the other two groups, but it does occur. Great care should be taken to prevent spilling of the contents of these cysts.

TREATMENT

The required treatment of all groups is operative, as an accurate diagnosis cannot be made without surgery, and operation should be advised early and insisted upon. The peritoneoscope should prove of inestimable value in making the diagnosis. It should be used by one accustomed to the instrument, and great care should be exercised not to perforate the growth. Just as the colposcope, the cystoscope and the proctoscope are of enormous value, so eventually may the peritoneoscope become.

Treatment should consist of radical surgery. Whenever possible both the ovaries, the uterus and the cervix should be removed. A good rule in cases of ovarian cancer is to remove as much tumor tissue as is possible, and also all the genital organs if the patient's condition permits. Because these various tumors are so apt to be bilateral, and because ovarian tumors may metastasize to the other ovary, both ovaries should be removed (Table 3). If possible a total hysterectomy should be done, for ovarian tumors can metastasize to the cervix by way of a chain of lymphatics in the uterine musculature. Certain schools advise leaving the uterus behind so that it can be used as a locus for radium therapy. Inasmuch as surgery is the best means of treatment and because the new x-ray apparatus can deliver lethal dosage into the pelvis, it is safer to remove the uterus than to leave it for subsequent radium treatment.

It is extremely important to open an ovarian tumor before finishing the operation, for papillary projections may be found within the cyst, and in these tumors papillary projections suggest malignancy. If such processes are found on the outside of the cyst it is best to adopt radical surgery and remove all the pelvic organs. If papillary projections are found in a freely movable tumor and if the patient is young and wants children, conservation is justifiable. In such cases it is important that the patient be seen at least every six months for five years or more, because if a neoplasm can develop in one ovary the same embryonic background is probably present in the other and a tumor may develop in it. Radical surgery is the rule and conservation of ovarian tissue the

TABLE 3. *Type of Operation in All Cases.*

TYPE OF CANCER	NO. OF CASES	PER CENT INOPERABLE	UNILATERAL OÖPHORECTOMY		BILATERAL OÖPHORECTOMY		BILATERAL TUMORS	
			PER CENT ALL CASES	PER CENT LIVING	PER CENT ALL CASES	PER CENT LIVING	PER CENT ALL CASES	PER CENT LIVING
Solid carcinoma	67	25	22	7	43	18	37	8
Malignant papillary cystadenoma	80	15	31	28	43	26	41	27
With adenocarcinoma	37	8	38	29	51	21	43	12
Without adenocarcinoma	43	21	26	27	35	33	40	41

exception in the proper management of malignant lesions of the ovary.

Before making a tremendous incision in the abdominal wall in the presence of a huge ovarian tumor it is often a great question as to whether or not it is safe to tap the cyst. The figures of this study (Table 4) and the previous one[1] show that statistically no harm comes from rupture or from tapping a cyst. This is probably due to the small number of cases, for the spilling of the contents of a papillary cyst usually spreads particles of tumor that are in the cyst fluid and this is liable to be harmful. In operating on other cancers, cutting across or spilling tumor is considered dangerous, and there is corresponding danger here. However, Hodenpyl[3] years ago advocated the treatment of cancer with ascitic fluid from patients suffering with malignant disease because he felt it was in some way inimical to cancer growth. It is possible that the fluid of these cysts may be an inhibitor of cancer growth. Nevertheless, it seems safer to avoid spilling and rupture if possible.

TABLE 4. *Miscellaneous Data.*

TYPE OF CANCER	ALL CASES		PER CENT RECURRENCE IN OTHER OVARY	UTERINE INVOLVEMENT		ADHESIONS		CYST RUPTURED BEFORE OR DURING OPERATION	
	NO.	PER CENT LIVING		PER CENT ALL CASES	PER CENT LIVING	PER CENT ALL CASES	PER CENT LIVING	PER CENT ALL CASES	PER CENT LIVING
Solid carcinoma	67	9	0	12	0	48	6	21	7
Malignant papillary cystadenoma	80	23	4	10	13	50	28	31	32
With adenocarcinoma	37	22	5	14	20	54	25	32	33
Without adenocarcinoma	43	23	2	7	0	47	30	30	31

Inoperable Growths. There were many inoperable cases in this series (Table 3); the patients were simply explored, a biopsy was taken and the incision was closed. In over a quarter of the solid-tumor group and in one seventh of the cystic group this was all that could be done. There were more patients in the former group who had a short history from onset to operation and more died in the first year after operation, showing that this type of cancer is definitely a more serious one than the cystic type.

Bilateral Tumors. In this series the survival rate of patients with bilateral solid tumors is below that of the entire group, but it is better in the cystic group (Table 3). Dividing the latter group into the two types, the patients with more malignant tumors that were bilateral fared worse than those with less malignant ones. It appears from this study that the patients with bilateral lesions had a more serious prognosis than did those with single tumors.

In our hospital figures the surgeons removed only one ovary in 30 per cent of the patients with operable solid tumors, 41 per cent of those with malignant papillary cystadenomas with adenocarcinoma in the wall and 32 per cent of those with malignant papillary cystadenomas. Thus, in spite of the fact that a high percentage are bilateral we failed to take advantage of our knowledge of ovarian tumors. It is a matter of interest, however, to find that only 5 per cent of the patients in the more malignant cystic group came back for removal of another tumor of the ovary, and only 2 per cent of those in the less malignant group, while none returned in the solid group (Table 4). All cases have been followed carefully for some time, so this fact is certainly interesting. It is probable that we have been more lucky than wise.

TABLE 5. *Operative Mortality and One-Year Mortality in All Cases.*

TYPE OF LESION	NO. OF CASES	OPERATIVE MORTALITY	ONE-YEAR DEATHS
		%	%
Solid carcinoma	67	10	64
Malignant papillary cystadenoma	80	4	58
With adenocarcinoma	37	3	59
Without adenocarcinoma	43	5	56

TABLE 6. *Five-Year Survivals in All Cases.*

TYPE OF CANCER	ALL CASES		PSEUDOMUCINOUS		ENDOMETRIAL		UNCLASSIFIED	
	NO.	PER CENT LIVING	PER CENT ALL CASES	PER CENT LIVING	PER CENT ALL CASES	PER CENT LIVING	PER CENT ALL CASES	PER CENT LIVING
Solid carcinoma	67	9	5	0	16	18	79	8
Malignant papillary cystadenoma	80	23	34	26	21	12	45	26
With adenocarcinoma	37	22	22	50	27	10	51	16
Without adenocarcinoma	43	23	44	16	16	14	40	35

OPERATIVE MORTALITY AND FIVE-YEAR SURVIVALS

In the solid group the operative mortality was only 10 per cent, and many of these patients were in poor condition (Table 5). In the two cystic

groups the mortality was 3 and 5 per cent, a fairly satisfactory result.

The results of surgical treatment, including the cases that had x-ray therapy, show that in the solid and the solid and cystic group only 9 per cent of 67 patients survived five years (Table 6). In the more malignant group of papillary cysts, — that is, those with adenocarcinoma in the wall of the tumor, — 22 per cent are living, while in the malignant papillary cystadenoma group 23 per cent have survived five years. The end results according to histological classification show that the most favorable types in the solid group are those of the endometrial type, with 18 per cent living; the pseudomucinous type was the worst. Taking the two cystic types together, there are 23 per cent of 80 cases living five years. Inasmuch as many of these cysts looked fairly benign, the end results are appalling. The unclassified and the pseudomucinous types did best, with 26 per cent in each group surviving five years, and the endometrial type did poorly, with only 12 per cent survivals.

It is well to consider the endometrial type. This tumor, like cancer of the endometrium, does not become very malignant until it has grown outside the ovary or uterus. Once free in the peritoneal cavity it is one of the most malignant of all pelvic tumors. In a series[5] of cases of cancer of the endometrium reported from the Pondville Hospital it was found that this tumor metastasized farther and more frequently than did cancer of the cervix and that once it was outside the body of the uterus nothing could check its growth.

The percentage of five-year survivals of malignant papillary cystadenoma of all types is 23; this is a reasonable figure for most malignant tumors. However, the survival rate for the solid types suggests that it is one of the most serious cancers of the human body.

TABLE 7. *Results of Roentgen Radiation.*

TYPE OF CANCER	NO. OF CASES	RADIATED		NON-RADIATED		PERIOD BETWEEN OPERATION AND DEATH	
		PER CENT ALL CASES	PER CENT 5-YR. SURV.	PER CENT ALL CASES	PER CENT 5-YR. SURV.	RADIATED	NON-RADIATED
						mo.	*mo.*
Solid carcinoma	67	45	7	55	14	28	16.7
Malignant papillary cystadenoma	80	41	21	59	23	32.6	41
With adenocarcinoma	37	46	18	54	25	27.6	34.5
Without adenocarcinoma	43	37	25	63	22	37.5	47.5

ROENTGEN-RAY TREATMENT

There can be no doubt of the therapeutic value of deep radiation, whether given with a 200,000-volt or a 1,000,000-volt machine, but so far it has not been shown in any large group of cases that it should in any way replace surgery. Whatever can be removed surgically should be, and it should not be assumed that if all the tumor cannot be taken out roentgen-ray treatment will care for the rest. It will prolong life but will not cure cancer. The changes in tumors observed following x-ray treatment in large numbers of cancers of the cervix are most convincing. Roentgen radiation will slow up the growth of cancer deep in the pelvis, both in the gross and microscopically, but it will not cure it. The present method of treating metastatic ovarian cancer can be improved upon, for although it is necessary to treat the entire abdomen, the usual procedure is to treat only two or three fields. This treatment cannot adequately cover the entire abdominal cavity, so that unless each field is marked off and treated, certain areas are sure to be missed. It is very important to insist on adequate and thorough radiation if it is to be given. Perhaps better results will come following the use of high-voltage machines, but this cannot be decided as yet. X-ray treatment should be given in all cases where tumor tissue has been left behind or where there is such a possibility, and it should be given as a prophylactic treatment to those patients in whom a malignant cyst has been ruptured and its contents spilled in the abdomen. If a clean and perfect removal has been accomplished it is safe to omit radiation, but it can be given in these cases also.

The end results of cases of cancer of the ovary following radiation after surgical removal are not well known; although our group is not a large one, it was well followed and studied, and the treatment was the best that could be given at that time in the Massachusetts General Hospital. Most of the cases were treated with a 200,000-volt machine with the usual screening, amperage, and so on. The treatment varied from time to time as it has in other clinics. It is fair to assume that these patients were adequately treated so far as treatment was possible. Undoubtedly better results have been obtained from those treated in the past five years, but it is of no use to report on them at this time. The complete details of radiation

treatment lie outside the scope of this paper. The gross results obtained by radiation in about 40 per cent of all the cases of cancer of the ovary reported are shown in Table 7. An important figure is that of the radiation group of solid cancers, with 7 per cent living five years without disease. This must be contrasted with a salvage of 14 per cent in a similar group not radiated. In the malignant papillary cystadenoma group the radiated cases did better than the non-radiated ones, but in the group with adenocarcinoma in the walls of the cyst, the non-radiated cases did better than the radiated. However, there is not much to choose between a 21 per cent salvage of radiated cases in the combined group and a 23 per cent salvage of non-radiated cases. The groups were fairly comparable, there being inoperable cases in both groups, and cases in which death from cancer occurred six to fifteen years later.

It is apparent from this study, so far as it goes, that no more people are cured with radiation than without it. Up to the present time the greatest reliance should be placed on surgery. It is more hopeful, however, to view the length of life in months of these patients having radiation as compared with those not having it, and here it is clear that in the most malignant type, the solid tumors, radiation prolonged life, but in the cystic groups life was longer without it. The whole series shows that the treatment given was not of too great value. It is my belief, however, that much better results will be forthcoming, and that further analysis of this particular group, with due consideration given to the extent of disease and the amount and type of radiation, may show that radiation of ovarian cancer is of greater value than now appears. But, without picking cases, this type of therapy has not proved so valuable as we thought it would.

DISCUSSION

Cancer of the ovary is a very serious lesion, and the solid type rates with the very worst of all malignant tumors. The record of our hospital is not satisfactory, but it probably represents the results in the community at large. Comparable series must eliminate all questionable tumors, must rule out all special tumors and must make sure that no non-malignant papillary cystadenomas are included. This series has been carefully studied and each case accepted only after careful consideration; it presents a gloomy picture, but I believe it is a correct one.

If found early, cancer of the ovary is curable, for it is often encapsulated in the ovary and is not serious until the cyst is broken or perforated or the tumor has grown through. Therefore early operation is essential in patients with questionable ovarian lesions. It is far better to remove a simple cyst or a fibroid because of a mistaken diagnosis than to wait to see whether a given lesion becomes malignant. The use of the peritoneoscope must be encouraged, and when it plays a more prominent part in the diagnosis of pelvic disease I believe that our figures will improve. The preservation of ovarian tissue in women with ovarian cancer or papillary cysts — benign or malignant — is serious, and if this is done in order to allow pregnancy to occur, extreme caution must be exercised in the follow-up, care being taken that a lesion does not start in the other ovary.

Tapping of cysts is probably not a sound procedure, but the figures in this paper do not prove it; in fact they suggest the contrary. It is safer to advise against tapping, but if a cyst is ruptured the surgeon should not give up hope, as he has the figures of this series of cases and Hodenpyl's[4] advice as precedents.

SUMMARY AND CONCLUSIONS

Cancer of the ovary of the solid type is a very serious neoplasm.

Cancer of the ovary of the malignant papillary cystadenoma type is about as malignant as any other epithelial growth.

Early diagnosis and methods with that in view are necessary to improve the end results of this easily operable tumor.

The use of the peritoneoscope should prove of great value in diagnosis.

Bilateral tumors are more serious than unilateral ones.

Bilateral oöphorectomy with total hysterectomy is the operation to be carried out if possible.

Postoperative mortality is low.

The rupture of cysts before and during operation and the use of the trocar cannot be proved dangerous by our end results; nevertheless, avoidance of spilling of cyst contents is advocated.

X-ray treatment to date has not proved of much curative value, but more modern methods of treatment may give greater success.

Every cystic or solid tumor of the ovary that is removed should be opened before the surgeon ends his operation, in order to rule out the presence of any suspicious papillary area.

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Discussion

Dr. Oliver N. Eastman, Burlington, Vermont: I congratulate Dr. Meigs on the thoroughness of his study and his able presentation of this important subject, which I believe has received too little consideration in recent literature.

Dr. Meigs has brought out the fact that the mortality is very high in ovarian malignancy. This can be accounted for largely by the fact that the symptoms of ovarian malignancy are usually not in evidence until the disease is well advanced. It may be stressed that ovarian tumors are not infrequent, and that the results of treatment will be better when the condition is recognized early and radical treatment instituted.

A pelvic tumor the size of a fist, especially if nodular and associated with irregular bleeding or ascites, warrants surgical investigation. On opening the abdomen, if a warty or papillary growth is noted on the surface of the tumor, radical surgery is indicated.

The ovarian tumors removed in our hospital during the last five years numbered 300. Twelve per cent were essentially or potentially malignant. There were 8 cases of papillary cyst adenocarcinoma, 4 cases of pseudomucinous cyst with malignant involvement and 1 case of malignant teratoma in a child of six. The other cases were questionably malignant. I believe that radical surgical intervention is the treatment of choice in all growths with papillary manifestations, regardless of the histologic characteristics. It is our custom to radiate following the removal of tumors which are suggestive of malignancy.

Will Dr. Meigs tell us how he takes care of omentum "cakes" so frequently associated with malignant tumors of the ovary?

Dr. Benjamin H. Alton, Worcester: Dr. Meigs's paper is to be commended for several reasons. First, it presents a large series of primary malignant epithelial tumors of the ovary from which have been sorted out those tumors which either clinically or histologically were considered benign or doubtfully malignant. The second point is the method he used in classifying the tumors both grossly and microscopically. A third has to do with the management of these tumors relative to lowering the mortality rate and a frank discussion of certain methods in the treatment of the primary malignant epithelial tumors of the ovary.

This series includes 147 cases, the choice from more than 250 tumors of malignant types, which represents more truly the primary malignant tumors that may arise from the epithelium of the ovaries. To include such tumors as embryomas, granulosal-cell tumors and a number of the cystadenocarcinomas which may be questionably malignant, and which were included in the series of cases reported by Lynch and Murphy, would lessen the value of a study of the real malignant tumors which are responsible for the large number of deaths.

We have observed that the mortality rate in external cancer, such as carcinoma of the breast, lip or skin, has been materially lowered through early recognition and radical removal by surgical means, or obliteration by x-ray therapy.

With reference to the early recognition of internal cancer, such as cancer of the ovary, Dr. Meigs has emphasized certain symptoms and physical findings which are of value. He has also emphasized that the age in which to suspect a malignant tumor of the ovary is between thirty and fifty, the peak being between forty and fifty. The relation of fertility to malignant disease of the ovary is extremely interesting, and in spite of the fact that 64 per cent of the patients in this group of solid carcinoma were fertile, we cannot infer too readily that sterility is a factor in the production of cancer of the ovary.

In the second group of patients, those having malignant cysts, he shows that the symptoms and physical findings are nearly parallel with those in the solid carcinoma. I believe that we can suspect malignancy of the ovaries sufficiently in cases presenting the above symptoms and physical findings to justify a laparotomy. I agree in principle with Dr. Meigs that it is better to remove a benign tumor of the ovary in doing an exploratory laparotomy than to wait until the more classic symptoms of malignancy have developed. As in carcinoma of the breast, we have learned that by the time these symptoms have developed surgical care is practically impossible. I am firmly convinced that any patient between the ages of thirty and sixty presenting the symptoms mentioned demands an exploratory laparotomy, not only as a possible life-saving measure but as a duty on the part of the surgeon.

It is to be understood, from a pathological point of view, that there are only two ways in which a cancer of the ovary may be spread to other structures. One of these is through implantation of tumor fragments; the other is via the lymphatics and blood vessels and along the fascial planes. Before a papillary process or fragment of tumor can appear on the surface of an ovarian cyst, the tumor cells must have passed from the lining of the cyst through the wall and peritoneum. At first, or early, they are small granular excrescences which may occur on a localized area or in blotches over the surface of the malignant cyst. It seems to me that when the surface of an ovarian cyst has a grayish frosting or appears granular in blotchy areas, we should recognize that this conforms with one method of spread of the tumor cells and malignancy should be strongly suspected. Not only this, but care should be exercised in handling these tumors for fear of rubbing off papillae or granules and losing them in the peritoneal cavity.

Because of the possible spread of the tumor through the lymphatics, blood vessels and fascial planes, our exploration should include an examination of the mesosalpinx, the mesovarium, the round ligament and the broad ligaments. If there is induration or thickening of one or more of these structures at the base of the tumor, we also know that this conforms to the spread of malignant disease and that the suspicion of cancer should be very strong. I believe that when these conditions exist, the general peritoneal cavity should be walled off and the entire internal genitalia removed. A metastatic nodule in the groin or lumbar lymph nodes indicates that the cancer is inoperable.

It is unfortunate that we have only one tool in our armamentarium that is effective in cancer of the ovary. This, as implied, is radical early surgery. Late surgery is only palliative. Perhaps one reason why these tumors of the ovary are less sensitive to radiation is that they are very often highly differentiated. It is well known that the embryomas and other tumors less differentiated, such as the granulosal-cell tumors, are more sensitive, but even these may recur.

We had at the Worcester Memorial Hospital, from 1929 to 1937 inclusive, 50 cases of primary epithelial tumors of the ovary. This, of course, includes a number of benign tumors such as papillary cystadenoma and multiple cystadenoma. At my request, the sections of these cases were reviewed by our pathologist, Dr. James P. Beck, with the idea of separating the definitely malignant tumors from this group. This was done, and it was found that there were only 13 malignant tumors. The criteria used in determining the malignancy were areas of adenocarcinoma in the wall of the cyst, mitotic fig-

ures, undifferentiation and atypicality of cells. It is seen that, of this group, only 26 per cent of our primary tumors are malignant. This is a much smaller percentage than the 55 per cent which Dr. Meigs has found in his group of 250 cases. A reason for this discrepancy may be that we separated the malignant tumors from the epithelial cysts and primary tumors which occurred in the ovary, while Dr. Meigs excluded the frankly benign ones before the series was compiled.

The outstanding points which Dr. Meigs has striven to make clear to us are the early recognition of carcinoma of the ovary, exploratory laparotomy and early radical surgery.

Dr. James R. Miller, Hartford, Connecticut: I have been going over the material at the Hartford Hospital since 1916 and no one who has not delved into material of that kind appreciates what an enormous amount of work Dr. Meigs has put together in this study.

One point that he did not touch upon, which is perhaps outside of his paper, is a reference to sarcoma of the ovary. It has been definitely shown that most of the patients with so-called sarcomas have actually had granulosal-cell tumors, and under that diagnosis have been cured of a benign lesion. We have, however, had a large number of granulosal-cell tumors which have been malignant; nevertheless, I think it is quite proper to take out all these differentiated tumors from the group of ovarian carcinomas.

Dr. Edward H. Risley, Waterville, Maine: I should like to ask Dr. Meigs if he will not state his attitude toward postoperative radiation of these tumors.

Dr. Meigs (closing): I believe that if the omental "cake" can be removed successfully and easily, and if there is no evidence of extension of the growth over the parietal peritoneum, it should be removed; or if the surgeon thinks that he has the tumor all out except that in the omentum, it ought to be removed; otherwise, I do not believe it is worth while.

To answer Dr. Risley's question about x-ray treatment, I believe that it will eventually prove to be of extreme value. Patients who have cancer of the cervix at the Pondville Hospital all have x-ray treatment before they are given radium. I have been studying the slides after that treatment, and there is no question but that this treatment changes the tumor remarkably. We have found that if tumors show radiation reaction in the stroma and in the epithelium of the cancer following x-ray, the chances of cure are good; but if the tumor shows no microscopic evidence of change, the chances are very poor.

I know that x-ray treatment can change epithelial lesions in the cervix, and therefore it ought to be used in epithelial growths in the peritoneal cavity. My objection is to the method of treatment, and I believe that roentgenologists will soon be able to deliver diffuse and widespread radiation so as to cover most of the abdomen. The surgeon should show the radiologist where the tumor masses and their extensions are. The surgeon takes out a tumor and says there are peritoneal metastases, and the x-ray department irradiates the abdomen. It is up to us surgeons to talk the proposed treatment over with the roentgenologist and see if a method cannot be established whereby the abdomen can be more fully and completely treated. We should be able to salvage a few more of these patients.

LIPODYSTROPHIA FACIALIS*

Case Report

Rubin Guralnick, M.D.† and Hyman Green, M.D.‡

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IN REPORTING 6 cases of lipodystrophy, Parmelee[1] reviews the literature extensively up to the time of his report; according to him, Coates[2] in 1925 summarized 63 such cases up to that year. The latter defines lipodystrophy as a disease characterized by a loss of subcutaneous fat from the face and upper portion of the body without any apparent ill health. He states that the disease affects children chiefly between the ages of five and eight, the ratio between girls and boys being 2:1. Parmelee states that the chief characteristics in his 6 cases were the thin, cadaverous facies and emaciation of the upper trunk, while the rest of the body appeared either normal or better than normal. There was no assignable cause for the malady in any of his patients, and the general health of all was very good. The children, being rather young, displayed no abnormal mental complexes as a result of their abnormal appearance. Parmelee states that there is a multiplicity of theories to explain the local character of the fat absorption, but that these only serve to emphasize the lack of knowledge as to the etiology.

Campbell[3] concurs with Coates in the definition of the disease as well as in its main characteristics. In his series of cases, also, girls were predominant. According to him, the loss of fat in girls is peculiarly confined to the face and trunk as far down as the iliac region, while the breasts and the region below the ilia show a well-defined hypertrophy, as though compensatory in nature. In boys, on the other hand, the characteristic loss of subcutaneous fat seems to be confined to the face only, the rest of the body remaining normal. He reiterates that the general health of the patient seems to be remarkably unaffected, and rules out the possibility of a pituitary disturbance's being the etiologic factor, because of the curious limitation of the ab-

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sorption of fat to well-defined areas. It would not be possible, he argues, for the anterior portion of the pituitary body, which apparently has to do with the control of fat distribution, to be so selective in its control as to affect only certain areas of the body surface. He cites a case in which, besides the characteristic appearance peculiar to the disease, the patient exuded an abnormally strong body odor, and from this he argues that an overactivity of the sebaceous glands of the face and trunk may have something to do with the peculiar loss of fat in these parts only. To substantiate his theory, he calls attention to the fact that the sebaceous glands of the lower portion of the body are, as is well known, very inactive, and that as a result of such inactivity this part of the body is not affected in this disease.

Thannhauser[4] in his work on lipoid diseases calls attention to Simons's disease (not to be confused with Simmond's disease), which is characterized by the localized fat absorption just described, and which Simons[5] himself called lipodystrophia progressiva. In 1911 Simons reported a number of such cases without known etiology; Meyer,[6] in reviewing Simons's cases, was inclined to believe that the etiology lay in a segmental trophoneurosis, although he could demonstrate no local changes in the nerves. He described a case of a woman who appeared very emaciated facially, yet whose breasts and the lower portion of whose body seemed in better condition than the average.

From this it may be seen that although the writers mentioned all agree in the general description of the disease, the etiology nevertheless remains obscure. This makes it rather a distinct entity, and on closer examination of all the facts presented, one should find no difficulty in differentiating this disease from the following:

Simmond's disease: a syndrome of hypophyseal cachexia with a train of symptoms quite distinct from any other disease.

Facioscapulohumeral muscular dystrophy: characterized by a wasting of the muscles but not of the fat tissue, and quite progressive in character.

Progressive muscular atrophy: differentiated along the same lines, and chiefly familial in character.

Progeria: senile appearance in children, together with other signs of senility which are very distinct.

Malnutrition and cachexia: a lack of adiposity in general, least of all in the face, the underlying cause of which can usually be easily detected.

Case Report

D. P., an 8-year-old girl, was brought to the Out-Patient Department of the Beth Israel Hospital on February 25, 1938, because of "thinness of face." Her present illness had begun when she was 4 years old, and it had become increasingly noticeable that her face was getting thin. This was more apparent because up to the age of 4 she was considered to be a rather plump child (Fig. 1). Her sister could not recall any unusual illness preceding this sudden change in the child's appearance, nor was there any noticeable change in the child's behavior, habits or appetite. It was also observed that while the face was getting thin it also took on a peculiar whitish appearance. The patient's condition continued to become progressively worse for 2 years, and on the advice of the family doctor a tonsilloadenoidectomy was performed. Since then there had been no further change in the facial appearance. Her appetite remained good and she got an adequate and fairly well-balanced diet. Her habits were

Figure 1.

good, and she generally got about 11 hours' sleep. She was active, helped with the housework and took part in the usual outdoor activities of children. She displayed no weakness on walking or during play, and got along well with her playmates. She did not seem to suffer from any complexes due to her facial appearance and was mentally alert, and her school rating was good.

The patient was the tenth of a family of eleven children. She was born at full term and of normal labor. At birth she appeared healthy and of average weight. She was breast-fed for a short while, and was then put on a Dextri-Maltose-milk formula. The addition of solid foods followed in due course. She had always had a sufficient amount of cod-liver oil and orange juice in infancy, and so far as known her development was not out of the ordinary. She walked and talked at the expected time and was considered an average healthy child. She had had measles, chickenpox and whooping cough before she was 2 years old. She also had had a few "slight colds." At the age of 6 her tonsils and adenoids were removed because of her appearance, as previously stated. She entered kindergarten at the age of 4 and had been regularly promoted.

The mother died of pneumonia at 41 when the patient was a little over 1 year old. The father died 2 years previous to admission at 52, of a tumor of the stomach. One

brother died of accidental drowning, and another of pneumonia in infancy. There were no miscarriages. There were four brothers and four sisters, all apparently in good health except one brother, who was totally blind as the result of an accident. The family lived in a tenement-flat cared for by the oldest sister, assisted by a state agency. The sleeping quarters were adequate and clean, and the food seemed to be adequate. There was no history of tuberculosis and none of allergic or mental disease. All the brothers and sisters were normal in weight and appearance, and there was no record of muscle dystrophy in any member of the family. The father and mother both came from Italy and married in this country, and all the children were born in or around Boston.

The patient was 49½ inches in height and weighed 60 pounds, a little above the average for her age. She was

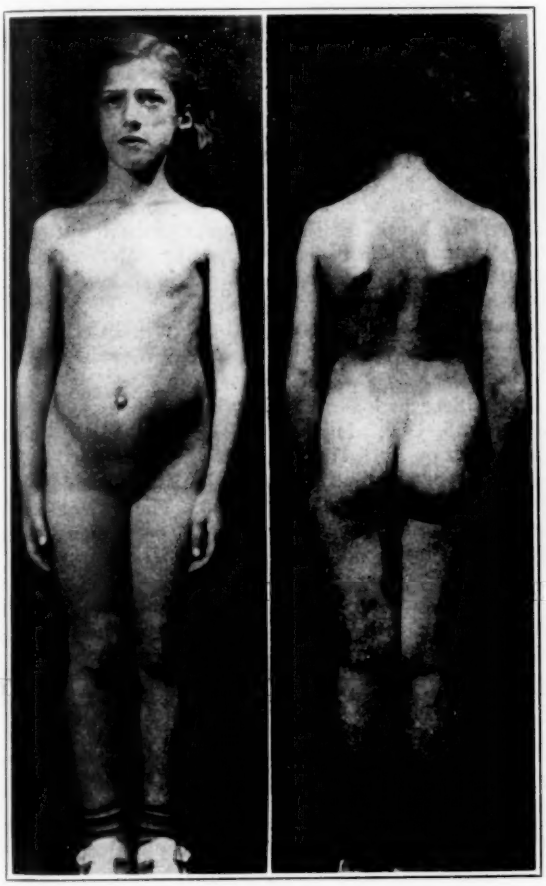

Figure 2.

bright and co-operative. The striking thing about her appearance was the drawn, thin face, with hollow cheeks and a peculiar pallid hue. The cheekbones were prominent and the skin lay in deep folds along the corners of her mouth, giving her a senile appearance. This thinness of face extended to the neck and slightly below the clavicles. On palpating the face, one was impressed by the thinness of the skin and the total absence of fat beneath it. When the patient was told to pucker her mouth the masseters stood out as small, thin bundles without any roundness of the surrounding tissue. On smiling, the skin wrinkled while the muscles around the mouth stood out grotesquely as in very debilitated old people.

The rest of the body appeared rather plump, particularly the pectoral regions and from the pelvic girdle down. The secondary sex characteristics, however, showed no precocity, and with the exception of somewhat poor dental development and slight icteric tinge of the sclera the rest of the physical examination appeared to be negative. (See Figs. 2 and 3.)

The red-cell count was 4,280,000, and the hemoglobin 75 per cent. The white-cell count was 8600, with 52 per cent polymorphonuclears, 38 per cent large lymphocytes, 8 per cent small lymphocytes, 1 per cent eosinophils and 1 per cent basophils. The achromia was 1+. The blood Wassermann and Kahn tests were negative. The blood cholesterol was 173 mg. per cent and the blood calcium 10.5 mg. per cent. The nonprotein nitrogen was within normal limits. The basal metabolic rate was +2 per cent. The sugar-tolerance test was as follows: fasting 74 mg., first hour 111 mg., second hour 77 mg. and third hour 47 mg. per cent. The urine was negative. An x-ray

Figure 3.

of the skull showed no abnormalities of sutures or sella, and an x-ray of the ossification centers showed development consistent with the patient's age.

SUMMARY

This case of lipodystrophia facialis in an eight-year-old girl is of interest because of its comparative rarity, as well as for the ease with which it may be overlooked. As may be seen from the description of the case itself, as well as from the brief review of pertaining literature, the condition is generally characterized by a gradual disappearance of the subcutaneous fat from the face and trunk down to the pelvic region, with some hypertrophy of the fat of the rest of the body. The general health remains remarkably unaffected. The disease affects children chiefly between the ages of four and eight and usually becomes stationary at about that age. Girls are the chief victims of this peculiar disease. The etiology is

unknown and the theories are conflicting and not convincing. Wasting diseases can be differentiated from it by the finding of a definite etiologic factor. Systemic treatment is of no avail. An increase in diet results only in an increase in the total avoirdupois without in any way changing the patient's appearance. The cosmetic correction of the peculiar facies by injection of paraffin into the subcutaneous tissues is not advisable because of the danger inherent in the use of a foreign substance.

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TRANSFUSION SYPHILIS

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THE performance of a blood transfusion without previous physical examination and a blood test for syphilis, although plenty of time in which to do them is usually available, seems to be a common practice. More than once I have questioned patients about blood tests, and have received answers such as the following: "I never had a blood test, but my blood must be all right since I gave it to a friend a few months ago. I was only typed and there was no emergency."

If this subject is brought up in private conversation among doctors, it is generally admitted that everyone knows of transfusion syphilis, but there is an evident desire to avoid having the fact become public.

The case of transfusion syphilis here reported is presented in order to call the attention of physicians to this matter. It shows what narrow escapes there may have been in the past from the accidental transmission of syphilis, in cases where reliance was placed on a donor's statement of good health or on a Wassermann test taken months before and so absolutely worthless. Klauder and Butterworth[1] investigated the medicolegal aspect of the subject and found that verdicts had been returned against doctors who made transfusions without previous serological tests for syphilis.

CASE REPORT

A 13-year-old boy examined on April 6, 1938, presented a maculopapular rash consistent with secondary syphilis. His blood Wassermann reaction was 4+ with both antigens. The approximate time of onset of the rash was the 1st week of March, 1938. It was not noticed whether it appeared first on the legs or on the body. It eventually appeared on the entire body and remained evenly distributed.

According to the patient's mother he was operated on November 30, 1937. The history showed that he was in a state of extreme cachexia, so that a fatal termination was momentarily expected. On December 2 he received an emergency blood transfusion (citrated) from a 20-year-old friend of the family. The donor's blood was typed but he was not given a physical examination. He was carrying a penile sore at the time of the transfusion. About January 1, 1938, he developed secondary manifestations of syphilis and gave a positive Wassermann reaction. He apparently never showed a rash. The chancre persisted for 6 weeks. At the end of April, 1938, he was symptom-free, but still gave a positive Wassermann reaction.

The patient had a negative Wassermann reaction on December 8, 1937. His mother and father each had a negative test on April 11, 1938. Toward the end of March, previous to any antiluetic therapy, he was up and about and had gained 40 pounds in weight. The patient's rash responded quickly to treatment with neoarsphenamine, administered intravenously.

From the data given above one can hardly doubt that syphilis was transmitted with the transfusion, which had been given without questioning the donor about sexual exposure, and without giving him a physical examination or a serological test for syphilis. In this case, however, even if a Wassermann test had been done on the donor's blood it might have brought a negative response, since the donor may have been in the preserological stage of infection. Moreover, even if previous to the transfusion he had been questioned about sexual exposure he could have denied it, and on examination what remained of the chancre might have escaped observation. The only conclusion that can be reached in this case, strange though it must appear, is that a pint of fully active syphilitic blood put a dying patient on his feet.

The infectiousness of syphilitic blood was reported for the first time in 1862.[2] To solve the problem of the transmissibility of syphilis by the blood, then under debate, three Italian physicians

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submitted to inoculation with syphilitic blood. After the incubation period one of them developed secondary manifestations. He died in 1922.[3] The other two escaped infection.

In order to ascertain how blood transfusions were being performed throughout the country, Levine and Katzin[4] sent a questionnaire to about seven hundred hospitals. Only three hundred and fifty of these responded, and many questions were answered vaguely. An inquiry as to the percentage of cases in which a serological test for syphilis was done previous to transfusion was unanswered by one hundred and thirty-six hospitals. Ninety-one stated that such tests were regularly omitted, reliance being placed on a routine test performed about every six months. Information concerning examination of the donor was unfortunately not covered by the questionnaire. According to Rein et al.,[5] in 2 per cent of the hospitals in the United States donors report for a blood test only once a year. Only 3 cases of syphilis were reported among the accidents following transfusion — obviously an unreliably small number. Rein et al. stated that the known cases of transfusion syphilis numbered 68.

Syphilis from transfusion is unquestionably a most deplorable accident. That there should be such a large number of cases, a fact which is not published but is well known, is without justification. As is true of syphilis contracted in other ways, the eradication of transfusion syphilis is impossible, but its frequency can undoubtedly be reduced to a minimum. To this end, all cases should be punctiliously reported instead of being consigned to a pious oblivion. Health authorities should give wide publicity to such cases among the members of the medical profession, and should warn them periodically of the possibility of transfusion syphilis, reminding them that even the existence of an emergency is not a valid excuse for their occurrence, since a Kline microscopic test can be done in an hour and a Hinton test in one and a half hours. Those in charge of operating rooms should be requested to keep in view and attached to transfusion apparatus reminders such as the following: "Have the donor and the recipient been given a blood test for syphilis today? Has the donor been questioned and carefully examined for syphilitic manifestations?"

Just as on the highway good brakes and proper operation of an automobile represent a very small accident hazard, so a properly performed blood transfusion should represent a minimum hazard. Accidents will happen, but they should be limited to cases where it is impossible to avoid them.

SUMMARY

A case of transfusion syphilis is reported, in order to stress the need of serological tests and careful examination of the donor for syphilitic manifestations immediately before every performance of blood transfusion.

The posting of warning notices in operating rooms is suggested in order to reduce the incidence of transfusion syphilis to an unavoidable minimum.

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DEATH IN NEWBORN AND STILLBORN INFANTS*

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IN a small series of 25 cases of death in infants, varying in gestation age from five to nine months, and in which there were available the postmortem material and clinical data, it was found that the cause of death in 64 per cent was traceable to intrauterine disturbances other than those related to the fetus or to difficult delivery. This formed the largest as well as the most interesting group. The remaining 36 per cent consisted of congenital malformations incompatible with extrauterine life (12 per cent), injuries due to difficult labor (20 per cent) and syphilis (4 per cent). While the number of cases is small, the findings seem to indicate accurately where the mortality of infants of this age is generally highest.

Along with the postmortem material a study of the changes in the placentas of most of the cases has been made, and correlated with the clinical data of the prenatal and confinement records. This correlation has been stimulating, and has afforded a better understanding of the anatomic findings in the placentas and bodies of the infants. Deaths due to congenital anomalies and injuries sustained during difficult labor and due to specific infection‡ will not be discussed. They are summarized in Tables 1 and 2.

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‡The single case of syphilis does not appear in the tables. The spirochetes were found in the ascitic fluid by dark-field illumination and in the tissues by Levaditi's stain. The blood Hinton tests were positive.

MATERIAL

The tissues consisted of the postmortem material accumulated in the past fourteen or fifteen months from cases admitted to the Memorial Hospital, and from others coming into the laboratory. The cases were taken in chronological order. Most of these infants were born in the hospital, so that the prenatal and confinement histories were readily available. A few were admitted to the hospital from immediately outlying districts, shortly after birth. Their histories, though adequate, were brief. One of the infants having multiple congenital anomalies (Case A38-7, Table 1) was an outside case possessing medicolegal interest, the body being brought to the laboratory for postmortem examination. The small series therefore includes only postmortem cases in which the cause of death was directly associated with pregnancy and labor.

TABLE 1. *Deaths Due to Congenital Malformations.*

Case No.	Clinical History			Anatomical Findings	
	Prenatal	Natal	Survival Time	Placenta	Infant
A38-6	Normal	LSA	3 hr.	Cysts of amnion; acute necrosis; yellow infarction.	Atresia of anus, ureters and urethra; polycystic kidneys (autopsy limited to abdomen).
A38-7	Unknown	Unknown	24 hr. (?)		Situs inversus; single-ventricle heart; meningoencephalocele.
A38-27	Toxemia, slight	LOP, 2 weeks before term	72 hr.		Hernia of diaphragm, left; hypoplasia of left lung; atelectasis of right lung; associated anatomy of asphyxia.

TABLE 2. *Deaths Due to Injuries Following Difficult Labor.*

Case No.	Clinical History			Anatomical Findings	
	Prenatal	Natal	Survival Time	Placenta	Infant
A37-26	Normal	Term, forceps; cord around neck.	48 hr.		Abrasion of skin; caput; atelectasis; hemorrhage into gastrointestinal tract; associated anatomy of asphyxia.
A37-31	Generally contracted pelvis	Term, LOP, version.	Stillbirth		Caput; tear of falx; intracranial hemorrhage; atelectasis; amniotic sac contents in lungs; associated anatomy of asphyxia.
A38-4	Rheumatic heart disease	1 month before term; ROA, Scanzoni.	8 days	Normal	Hemorrhage into meninges; massive hemorrhage into lungs; amniotic sac contents in lungs; atelectasis; associated anatomy of asphyxia.
A38-8	Normal	Term; LOA; forceps.	72 hr.		Abrasion of skin; tear of tentorium, left; massive intracranial hemorrhage; atelectasis; associated anatomy of asphyxia.
A38-17	Normal	2 weeks over term; LSP.	48 hr.	Normal	Atelectasis; marked hemorrhage into lungs; associated anatomy of asphyxia.

In Table 3 are grouped the 16 cases selected for discussion. Among them are 7 stillbirths (28 per cent) and 9 liveborn infants (36 per cent) whose postnatal life averaged nine and a half hours. The shortest postnatal life was fifteen minutes in a premature infant weighing 20 ounces. The longest was thirty-six hours in an infant weighing 5 pounds, 4 ounces. Clinically there were in every case objective prenatal symptoms pointing directly to a disturbance in the placenta or to the systemic diseases characteristic of pregnancy.

Ten of the 16 mothers had some type of tox-

emia. One had eclampsia and the remaining 9 showed albuminuria, edema, elevation of blood pressure, some retention of nonprotein nitrogen and varying grades of anemia. Seven cases showed the objective signs of a disturbance in the placenta or membranes, such as vaginal bleeding and premature rupture of the membranes. Three mothers each contained areas of infarction showing acute necrosis. The infant of the eclamptic mother was stillborn and the placenta showed no marked gross changes. There were two areas of hemorrhage each measuring about 2 cm. in diameter. The microscopic changes were those of infarction and acute necrosis.

TABLE 3. *Intrauterine and Extrauterine Deaths Due to Asphyxia.*

CASE NO.	CLINICAL HISTORY			ANATOMICAL FINDINGS	
	PRENATAL	NATAL	SURVIVAL TIME	PLACENTA	INFANT
A37-15	Toxemia	8½ mo. gestation; ROA; birth weight 88 oz.	10 hr.		Atelectasis; amniotic sac contents in lung; associated changes of asphyxia.
A37-19	Eclampsia; premature separation of placenta.	9½ mo. gestation; LOP; birth weight 104 oz.	Stillbirth	Infarction; acute necrosis.	Atelectasis; amniotic sac contents in lung; associated changes of asphyxia.
A37-20	Toxemia; vaginal bleeding; premature separation of placenta.	6 mo. gestation; LOA; birth weight 44 oz.	1 hr.		Prematurity; atelectasis; amniotic sac contents in lung; associated changes of asphyxia.
A37-27	Threatened miscarriage	6 mo. gestation; PP; birth weight 32 oz.	19 hr.	Placentitis	Prematurity; atelectasis; amniotic sac contents in lung; associated changes of asphyxia.
A37-28	Toxemia; premature separation of placenta.	9½ mo. gestation; birth weight 112 oz.	Stillbirth		Atelectasis; amniotic sac contents in lung; associated changes of asphyxia.
A37-32	Premature rupture of membrane	7 mo. gestation; LOA; birth weight 72 oz.	2 hr.	Emboli in veins?	Prematurity; atelectasis; associated changes of asphyxia; focal necrosis.
A37-37	Vaginal bleeding; premature separation of placenta.	8 mo. gestation; LOP; birth weight 80 oz.	Stillbirth		Atelectasis; amniotic sac contents in lung; associated changes of asphyxia; maceration.
A37-50	Toxemia	8 mo. gestation; birth weight 56 oz.	Stillbirth	Infarction; acute necrosis.	Prematurity; atelectasis; amniotic sac contents in lung; associated changes of asphyxia; maceration.
A37-53	Toxemia	7 mo. gestation; cesarean; birth weight 64 oz.	6 hr.	Normal	Prematurity; atelectasis; amniotic sac contents in lung; associated changes of asphyxia.
A38-3	Premature separation of placenta	8 mo. gestation; cesarean; birth weight 84 oz.	36 hr.	Normal	Atelectasis; amniotic sac contents in lung; associated changes of asphyxia.
A38-5	Toxemia	8 mo. gestation; ROA; birth weight 56 oz.; cord around neck.	Stillbirth	Deciduitis; sinus thrombosis; infarction; acute necrosis.	Prematurity; atelectasis; amniotic sac contents in lung; associated changes of asphyxia.
A38-9	Toxemia	7½ mo. gestation; breech; birth weight 40 oz.	Stillbirth	Infarction; acute necrosis.	Prematurity; atelectasis; associated changes of asphyxia; maceration.
A38-13	Toxemia	7 mo. gestation; LOA; birth weight 52 oz.	3 hr.	Deciduitis; sinus thrombosis; infarction; acute necrosis.	Prematurity; atelectasis; associated changes of asphyxia.
A38-16	Toxemia	7 mo. gestation; cesarean; birth weight 52 oz.	9 hr.	Placentitis; sinus thrombosis; infarction; acute necrosis.	Prematurity; atelectasis; associated changes of asphyxia.
A38-20	Premature separation of placenta	6 mo. gestation; birth weight 20 oz.	15 min.	Normal	Incompatible prematurity; atelectasis; associated changes of asphyxia.
A38-24	Premature separation of placenta	5 mo. gestation; birth weight 12 oz.	Stillbirth	Sinus thrombosis; acute necrosis.	Incompatible prematurity; atelectasis; associated changes of asphyxia.

showed both toxemia and vaginal bleeding in the late prenatal period. As a rule the labor was not difficult, and from the necropsy findings no evidence of trauma was discovered.

Three of the mothers of stillborn infants had toxemia without vaginal bleeding, but the placenta of each showed infarctions with acute necrosis. The mothers of the remaining 4 stillborn infants had vaginal bleeding before the onset of labor. Only two of these placentas were examined, and

The mothers of the 2 extremely premature infants (Cases A38-20 and A38-24) presented no symptoms of toxemia, but both had vaginal bleeding and showed evidence of premature separation of the placenta. One infant was liveborn, living for fifteen minutes postnatally, and the placenta showed no abnormal change, either grossly or microscopically. There were 6 liveborn premature infants. Three of the mothers had toxemia, 2 had vaginal bleeding and 1 had both.

In the 16 cases only 2 of the liveborn infants weighed 5 pounds or more. They were regarded as full-term. The mother of one had signs of premature separation of the placenta and the infant was delivered by cesarean section; the placenta was not remarkable either grossly or microscopically. The mother of the other infant had toxemia. The placenta was not examined.

DISCUSSION OF ANATOMICAL FINDINGS

The anatomical findings in the bodies of the 16 infants were simple, and consisted of varying degrees of prematurity, atelectasis neonatorum, and aspiration of amniotic sac contents, associated anatomical findings of asphyxia and varying degrees of maceration of body tissues.

Prematurity. Eleven infants (44 per cent) were premature by weight and measurement, but anatomically only 2 had lungs insufficiently developed to be compatible with life without placental circulation. One of these was stillborn; the other was a liveborn infant whose postnatal life was fifteen minutes. While prematurity may have been the immediate cause of death in the infant living for fifteen minutes, it could not have been the cause of death in the stillborn, although the anatomical findings were identical. (For comparison, see Cases A38-20 and A38-24.) Also the mothers of these two infants presented similar antepartum symptoms. The difference in the two cases lies in the timing of birth and in the placentas. The placenta of the stillborn showed an extensive pathologic change.

The primary cause of death may not be found in the bodies of premature infants. The search for a causative factor in such cases regularly includes an investigation of possible anatomic changes or other conditions responsible for premature birth. The cause of certain premature births may be the same as that producing intrauterine fetal asphyxia. The objective maternal symptoms of varying degrees of toxemia of pregnancy, vaginal bleeding or threatened miscarriage show that there were disturbances in the functioning of the uterus and placenta sufficient to cause death of the fetus either before delivery or soon thereafter.

Atelectasis and Associated Findings of Asphyxia. Every one of the infants in this series showed atelectasis. While its extent was not estimated it was observed that (except in the 2 cases of extreme prematurity) most of the alveoli were anatomically capable of inflation, and that generally the number of alveoli partially inflated was small in those infants who lived only a few hours after birth.

Clinically the infants were living in a state of partial asphyxia, and it may be assumed that the sustained asphyxia in part was due to a persistence of atelectasis. All newborn infants have atelectasis, and are normally endowed with a mechanism for overcoming it. This mechanism has been carefully studied by Wilson and Farber,[1] who have shown that the force necessary for the first effective inspiration is normally greater than all later ones. If the first inspiratory efforts are for any reason inadequate to inflate the first few alveoli, atelectasis will persist, and will continue to persist so long as this force is inadequate. The force of inspiration is a function of the respiratory mechanism involving anatomically the medulla oblongata, the phrenic and thoracic nerves, the muscles of the body wall and diaphragm and the bony structure of the thorax. Chemically it is affected as are other tissues by various injurious agents, and normally it is delicately sensitive to physical and chemical changes in the blood. In order for the mechanism to function it must be anatomically possible for very definite differentials to be established between the pressure in the thorax and abdomen and that of the atmosphere. The elements of the mechanism are recalled mainly for the purpose of showing the wide field in which abnormalities, largely responsible for persistent atelectasis, may be found in the bodies of newborn infants.

The bony defects and other gross anatomic abnormalities making impossible the establishment of adequate pressure differentials are determined by inspection and are congenital malformations.

It is well known that the rhythmic action and force of respiration are dependent on a normal neuromuscular reflex component and a normal chemistry of the blood. Traumatic injuries, inflammations and tumors of the brain and cord may weaken, alter or prevent the anatomical component from functioning. Muscular fatigue from prolonged ineffective inspiratory efforts in obstructed breathing results in a loss of force. These infants, without other assistance than removing the obstruction, may not be able to overcome atelectasis. The loss of muscular power associated with a severe anoxemia at the time of birth may result in respiratory efforts too feeble to start the process of alveolar inflation.

Important data concerning the chemistry of the blood of newborn infants are frequently reported, tending to indicate that there is a relation between the chemical constituents of fetal blood and those of the maternal blood. This implies that the principles of exchange of soluble substances are the same in the placental vessels as they are in the vessels elsewhere in the body. When bacterial

toxins, drugs, abnormal quantities of metabolites or alterations of the protein or mineral balance are present in the maternal blood some of the substances or alterations may be present in the fetal blood. A diminished carbon-dioxide combining power in the fetal blood (as inferred from a similar change in the maternal blood before labor) may be reflected in fetal muscular weakness and hence the respiratory efforts after birth may be weak. In the case of drugs the stimulative or depressive effect on nerve tissue is known. Hypnotics and sedatives reach the fetal blood through the placenta and may depress the respiratory center.

From the standpoint of morbid anatomy there are few lesions distinctive of such chemical injuries. Acute venous engorgement of the viscera with varying degrees of edema of the organs and tissues in general, occasional pleural and abdominal effusion, ecchymotic hemorrhages in the epicardium, pleura, thymus, meninges, peritoneum, and so forth, are the usual associated anatomical findings. Whatever the contributions to these changes are from the effects of drugs, toxins or certain injuries due to trauma, they cannot be precisely separated from those due to anoxemia. Death in utero or shortly after birth, due to asphyxia, is accompanied by anatomic changes indistinguishable from those described and defined as due to shock[2] experimentally produced by toxins, drugs or trauma. It is a common observation among obstetricians that those newborn infants who have "blue asphyxia" have an excellent chance of recovery, while most of those having asphyxia pallida soon expire. The latter are the ones that have the clinical outward appearance of shock and form the majority of cases for postmortem examination. There is a need for differentiative clinical laboratory data on these cases to separate clinical shock from asphyxia.

In the microscopic sections the pink-staining precipitate seen filling the alveoli of the lungs in experimental shock or asphyxia has its origin in edema fluid rich in proteins. Pink-staining precipitate is regularly seen in the partially inflated alveoli of the atelectatic lungs. The origin could be from edema fluid, but when the formed elements of the amniotic sac contents are present with it, some of the precipitate is undoubtedly due to the proteins of the amniotic fluid. Regardless of its origin, this fluid in the lungs produces a mechanical embarrassment to respiration. To withstand such anatomic changes demands a factor of safety in the cardiovascular-respiratory reflex mechanism. By this is meant that in non-atelectatic lungs, where the forces of congestion and edema are applied, the work of alveoli rendered functionless by the fluid is accomplished by greater work on the part of the normal alveoli through hyperpnea, orthopnea and increased pulse rate. There is little or no such factor of safety in the atelectatic lungs of the living newborn because there is no excess of ventilating space to be used for compensatory purposes. Trivial as they may seem in relation to lungs without atelectasis, such elements as small masses of mucus, amniotic sac contents or edema fluid in the respiratory passages may well cause enough obstruction in a newborn infant already suffering from asphyxia to prevent the feeble efforts of respiration from becoming effective.

While these embarrassments to the inflow of air may be a contributing cause of death, the outstanding pathological finding is the asphyxia which the infant had before birth was complete. If the anatomical parts of the respiratory mechanism are normal, it may be assumed that the obstructing elements in the bronchi and lungs are there because of a preceding asphyxia. The anatomical findings of asphyxia and amniotic sac contents in the lungs may place the onset of the abnormal changes in the period of intrauterine life. Similarly, vaginal secretion and blood in the respiratory passages may place the date at the time of delivery. Hence, the causative factor may be found in the placenta and uterus or be associated with delivery.

Aspiration of Amniotic Sac Contents. The determination of this condition is made by a microscopic examination of the lungs. The finding of lanugo hairs, large numbers of flat cells, and particles of vernix or granules of pigment or both (presumably from meconium) in the air spaces of the lungs simply indicates that the sediment of the amniotic fluid was aspirated before the amniotic sac was emptied. The importance of such findings has been questioned by some, and by others considerable importance is attached to them if associated inflammatory changes are present in the alveoli. Opinion differs as to whether these bodies can produce inflammatory changes, and it is considered by many that the inflammatory reaction in the lungs of the newborn is suggestive of aspirated vaginal secretion, particularly if bacteria are present. It is generally believed that the presence of large amounts of the sediment in the lungs is indicative of intrauterine asphyxia.

Theoretically there are two abnormal ways in which amniotic sac contents can be forced into the fetal respiratory passages. They may be forcibly aspirated as a result of an intrauterine agonal state during asphyxia, or may be forced into the lungs during uterine contractions. The latter con-

dition appears physically unlikely if the fetus is suspended in the unruptured amniotic sac, because the increased external fluid pressure due to uterine contraction is equally distributed throughout all parts of the fetus, producing no differential in pressure and therefore no movement of fluid into the fetus. Such movement becomes possible, however, in breech or foot presentations with delivery. The nose and mouth may still be in the uterine cavity while the remainder of the infant's body is in the birth canal. Amniotic sac contents, pooled blood or mucus can then be forced into the respiratory passages during uterine contractions, because in this case the pressure of the contracting uterus is directed against the intrauterine content, squeezing the material into the nose and mouth, while the abdomen or thorax is subjected only to birth-canal pressure.

In our series about 50 per cent of the infants, both stillborn and liveborn, had amniotic sac contents in their lungs. This subject has been clearly discussed in a paper by Farber and Sweet.[3] They found amniotic sac contents in 88 per cent of 124 infants who lived from two hours to five weeks. In their paper the relation of the aspiration of this material to intrauterine asphyxia is shown, and the importance of such aspiration as an additional cause of respiratory embarrassment of the newborn is emphasized.

It is well, however, in this discussion to point out that such elements as small masses of mucus or aspirated amniotic sac contents, meconium, vaginal secretion or blood in the respiratory passages of newborn infants may cause a persistence of atelectasis and asphyxia in two ways. One is the mechanical embarrassment to the inflow of air to those alveoli in the field of obstruction. The other, theoretically, is the presence of interstitial edema and pleural effusion which is partly due to anoxemia.

Clinically, in the event of such obstructions, the chest may diminish in size slightly, instead of expanding, during the inspiratory phase (when the diaphragm descends and the abdomen swells owing to the force of atmospheric pressure on the non-rigid chest wall). During the expiratory phase the chest resumes its normal size as the abdomen diminishes proportionately. The chest wall is normally not so rigid immediately after birth as it is a few days later. In premature infants the rigidity is proportionately less than that in term infants, making this ineffective paradoxical breathing more noticeable. Since the negative intrathoracic pressure is most marked immediately above the diaphragm as it descends, the Litten sign is usually pronounced in cases of obstruction.

Maceration of Body Tissue. This anatomic change is at times helpful in determining the approximate time of death before delivery of the stillborn. The degree of tissue maceration is more or less proportional to the time interval between death and delivery.

Anatomic Changes in the Placentas. It seemed at the outset that the placentas should be examined grossly and microscopically with the same thoroughness that any surgical specimen receives. This became especially apparent when it was realized that this organ alone was acting for the lungs, gastrointestinal tract and kidneys of the individual whose body was presented for postmortem examination and whose major clinical life was spent in utero while this organ was functioning. Also, emphasis was placed on the placenta-uterus relation by such objective prenatal symptoms as toxemia of pregnancy, vaginal bleeding, threatened miscarriage, and so forth.

The anatomic changes consisted of infarctions, areas of necrosis, acute inflammatory changes in and about the villi and thromboses of maternal sinuses. Grossly the infarctions were characterized by fairly well-circumscribed yellowish or white areas, varying in size but averaging about 2 cm. in diameter. Microscopically they were either masses of loose fibrin containing red cells and leukocytes, or were compact clumps of fibrillar hyaline material. Areas of necrosis are frequently associated with infarctions, resembling them in size but differing from them by having central cavitations which microscopically contain debris, leukocytes or poorly staining, partially autolyzed placental remnants. Many of these areas appear to be walled off by parallel strands of fibrin arranged in concentric fashion. Commonly a zone of compactly crowded poorly staining villi with pyknotic nuclei encircle these areas outside the wall of fibrin and leukocytes. Focal areas of necrosis may be found in the cotyledons, apparently unassociated with such infarctions and without a wall of fibrin. Sometimes the areas of necrosis consist of a mass of crowded necrotic villi seemingly held together by a coarse network of loose fibrin and leukocytes. When the villi show little necrotic change and are loosely arranged in the cotyledons, but contain neutrophils in their stroma and loose fibrin with leukocytes in the intervillous spaces, acute inflammation is implied. Inflammatory changes associated with small foci of necrosis and thrombosis of the maternal sinuses are seen in the decidua basalis. The frequency of these changes in the placenta and their relation to those found in the fetus may be seen in Table 3. These findings are few and

simple, making their value at present uncertain. It seems apparent, however, that there is a definite relation between these lesions and the abnormal physiologic changes causing death of the fetus in selected cases. The coexistence of eclampsia or toxemia of pregnancy and premature separation of the placenta with areas of acute necrosis is an example of the changes which lead to prematurity, persistent atelectasis and death in the fetus and newborn.

The clinical application of the disturbances in fetal circulation is in the evaluation of the grade of asphyxia in terms of fetal heart rate. Richardson[4] has evaluated the grade of asphyxia in varying degrees of premature placental separation by means of the fetal heart rate before and during delivery. He has shown that the fetal rate is 150 to 160 when one fourth of the placenta is separated prematurely, and when half the placenta is separated the rate reaches 170 to 190. When more than half is separated the heart begins to decompensate; the rate falling to 90 or 70 in the event that three fourths of the placenta is detached. This indicates that a drop in the rate after the rate has been rapid is an unfavorable sign. While Richardson's findings were related to premature separation, they indicate a principle applicable to embarrassment of fetal circulation in general. Such conditions as a tightly knotted cord, a prolapse of the cord and extensive infarctions destroying one fourth or more of the placenta usually affect the circulation and influence the fetal heart rate in a similar manner. Since the oxygenation of fetal blood becomes a function of the lungs after birth, the grade of obstruction to the inflow of air and the degree of atelectasis may be surmised from a similar alteration in the heart tones during the early neonatal hours. If the rate is slow immediately after birth it may indicate that decompensation is already at hand and that resuscitation procedures are immediately imperative. A few precious minutes of effective treatment at such a time may save the infant's life. On the other hand, if the process of overcoming atelectasis has begun and is progressive, there generally follows clinical improvement, and a gradual lowering of the heart rate in the course of a few days.

CONCLUSIONS

The usual anatomical findings following deaths of the fetuses in utero and liveborn infants are discussed with reference to etiology. Our finding of a close relation between death and the complications of pregnancy or labor or both is in complete harmony with a recently reported[5] relation involving many hundreds of similar cases. The pathogenesis of the anatomic changes is considered with respect to immediate and contributing causes of death. In the discussion a few clinical differentiative tests and observations are indirectly suggested for early recognition of the chief pathologic changes in the living newborn. It is to be assumed from the discussion that all ailing prospective mothers should be brought as near to normal health as possible before delivery is allowed to start if the welfare of the infant is the more important. The blood in anemia should be brought to normal range; in acidosis or alkalosis the carbon-dioxide combining power of the blood should be adjusted to normal; in nutritional edema the blood proteins should be raised; and the toxemias of pregnancy should be treated with great care. Mothers having a history of abortions, miscarriages, stillbirths or premature births should be given vitamins and suitable hormones in the prenatal period in order to carry the pregnancy to term. Narcotics and anesthetics seem contraindicated in mothers below par. Finally, it is to be expected that infants of such mothers will do badly immediately following birth, and that the armamentarium for immediate and effective removal of foreign materials from the respiratory passages should be ready for use. Effective means for resuscitation should be made available for immediate use during the critical period just after the cord is severed. It seems logical to cleanse the nose and throat before the cord is cut, and to use resuscitation measures afterward.

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REPORT ON MEDICAL PROGRESS

CHILDREN'S SURGERY

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IN THIS paper no attempt will be made to cover all the advances in children's surgery, but rather a few subjects with which it is thought the general surgeon or practitioner may be less familiar than is the surgeon giving particular attention to this age group. In some of these conditions progress has apparently been made by diverging from the usually adopted methods of procedure.

IDIOPATHIC PERITONITIS (METASTATIC PERITONITIS)

The term idiopathic peritonitis denotes an inflammation of the peritoneum due not to a spreading infection from inflammation of the appendix or fallopian tubes or from perforation of any of the abdominal viscera, but to a blood-borne infection involving primarily the peritoneal cavity. The organisms most commonly causing this type of peritonitis are the pneumococcus and the streptococcus. This condition is almost entirely limited to children, and perhaps, for that reason, is often inappropriately handled by those of limited experience in this age group. In recent years an attempt has always been made to differentiate preoperatively peritonitis of appendiceal origin and that of the idiopathic type.

This disease is characterized by an acute onset with abdominal pain and vomiting, and sometimes there is a history of an upper respiratory infection or diarrhea. The patient shows evidences of profound toxemia, prostration, a high temperature (104 to 105°F.), a rapid pulse, a high white-cell count, generalized abdominal tenderness and muscle spasm. Idiopathic peritonitis is differentiated from peritonitis of appendiceal origin in that the tenderness becomes more general earlier in the course of the disease, the temperature is higher than is usual in appendiceal peritonitis, the white-cell count is higher, and there is greater and earlier prostration.

During the last fifteen years papers have been published giving a mortality of 65 to 100 per cent in this condition. In 1926 Lipshutz and Lowenburg[1] reported a mortality of 100 per cent; in 1930 a series of 51 cases was reported from the Children's Hospital[2] in Boston with a mortality of 65 per cent; in 1934 Donovan[3] reported a 75 per cent mortality in pneumococcal peritonitis. During the period mentioned it has been a common practice to make a thorough exploration of the abdominal cavity for the origin of the peritonitis when it was found not to be the appendix. A good many years ago at the Children's Hospital it was recognized that this quest was in vain, and resulted not in the finding of the origin of the infection but in the speedy demise of the patient. We therefore adopted the policy of doing very little at the time of operation. The operation consisted solely in opening the abdomen, examining the appendix to make sure it was not the cause of the peritonitis and inserting a drain. This policy resulted in an improvement in results, so that in 1930 the mortality from both pneumococcal and streptococcal peritonitis was only 65 per cent. As a result of favorable reports on delaying the operation until such time as the localization might take place, we followed that policy for a number of years. This plan of treatment undoubtedly resulted in the saving of some lives that would have been lost by early operation. The drawback was that localization frequently failed to take place, and that many patients died during the period of delay. It must be recognized that in the child the length of the omentum relative to the size of the abdominal cavity is less than in later life, and for that reason, and perhaps on account of the greater activity of the child, localization of an inflammatory process in the peritoneal cavity takes place less frequently than in the adult. Delayed operation did not cause an improvement in our results — in fact the mortality rose to 72 per cent in a series of 54 cases. Recently we have swung back to early operation supplemented by chemical or serum therapy, as indicated. The present plan of therapy consists in making a small abdominal incision under novocain or nitrous oxide and oxygen anesthesia. As soon as the peritoneum is opened, the character of the pus and the appearance of the peritoneum make the diagnosis certain enough to allow the surgeon to avoid further exploration. Some of the pus is taken for the determination of the organism and a drain is inserted. The merits of the drain are, of course, open to argument, but from our clinical experience it is believed that it accomplishes some good in

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itself, and probably more in helping to localize the process.

As soon as the results of bacteriological examination are ascertained, suitable treatment is instituted. If the organism is a streptococcus, sulfanilamide is administered. If it is a pneumococcus of the type having an antiserum, this form of therapy is given. The aftercare of these patients is of far more importance than the operative procedure. Morphine should be given in doses sufficient to relieve pain and to prevent restlessness. Adequate amounts of parenteral fluids must be given in order to combat dehydration and ketosis, but excessive amounts should be avoided on account of the danger of circulatory embarrassment. In cases calling for the use of sulfanilamide a daily or bidaily blood examination should be made. It is common for this drug to cause a marked drop in the red-cell count and hemoglobin, which should be treated by transfusions. Abdominal distention is best relieved by Wangensteen suction and placing the patient in an oxygen tent with a high percentage of oxygen. The drain is removed when the temperature has reached normal, or a sinus tract has been definitely established, which is usually between the seventh and tenth days. This plan has not been followed for a sufficiently long time for us to feel certain of its merits, but our results so far are extremely encouraging. In 15 cases of idiopathic peritonitis treated in this manner, there were 3 deaths, a mortality of 20 per cent. Of these 15 cases, 8 were due to pneumococci and showed a mortality of 13 per cent, and 7 were due to streptococci and had a mortality of 29 per cent.

WILMS'S TUMOR (EMBRYOMA OF THE KIDNEY)

Embryoma of the kidney is a mixed tumor of great malignancy, of congenital origin, and usually manifests itself in the first two or three years of life. Clinically, these tumors seldom give symptoms other than the appearance of a rapidly growing mass in the region of the kidney. The general health of the infants is not affected even when the tumor has assumed enormous size. The urinary findings are only occasionally significant in showing red blood cells in the sediment, and pyelograms are seldom more than suggestive of the diagnosis. The latter, however, can be correctly made, with very few exceptions, on the basis of the history and the physical examination. These tumors cause an extremely high mortality, but we believe that we have made some progress at the Children's Hospital by diverging from the common practice of other clinics.

A recent extensive review of the literature reveals that it has been a common practice in recent years to irradiate these tumors for a period of three to six weeks prior to performing a nephrectomy. In a recent publication on progress in urology by Colby[4] and in one by Kerr,[5] it is implied that this form of treatment is an advance. Familiarity with the results obtained by it and without it make this implication controversial. It is agreed by all observers, so far as I know, that embryomas of the kidney are extremely radiosensitive, and that they decrease in size very rapidly as a result of x-ray therapy. It is also agreed that no patient with a Wilms's tumor has ever been cured by irradiation alone. Recent careful microscopic examination of irradiated tumors shows that some cells are apparently destroyed, while some remain viable. The breaking down of tissue which results from x-ray therapy leaves viable cells in a position to be taken up more readily by the blood stream. Clinically, it is suggested that metastasis takes place earlier in patients who have received x-ray therapy than in those who have not. Furthermore, it is impossible to determine the time at which metastasis takes place, or that it has not occurred during the period of regression under irradiation treatment. After a careful search in the literature last spring, I was able to find reports of but 8 probable cures of embryoma of the kidney in children from other clinics. Of these, only 3 had received preoperative irradiation, while 5 had received none. When the 11 cases recently reported by the writer[6] are added to the 5 reported by others, it shows that in 19 cases reported as probable cures, 16 patients had received no preoperative irradiation. It would seem that until such time as x-ray therapy becomes more successful immediate operation is indicated and offers the best outlook for cure. This statement implies, of course, that the operation can be done with a low operative mortality. In the last ten years at the Children's Hospital, 30 patients with kidney embryoma have been operated on, with two operative deaths, a mortality under 7 per cent, and no patient has been regarded as inoperable on account of the size of the tumor. It is believed that the factors contributing to improved results are careful preoperative preparation, avoidance of unnecessary delay and operative technic. A transperitoneal approach is a distinct advance over the previously used posterolumbar incision. The renal pedicle and ureter are tied and cut before the kidney is mobilized. This minimizes the possibility of liberating tumor cells into the blood stream at the time of operation. Parenteral fluids and transfusion are used postoperatively when indicated.

SURGICAL OBLITERATION OF THE PATENT DUCTUS ARTERIOSUS

Dr. Robert E. Gross at the Children's Hospital has opened a new field of surgical endeavor by operations on one form of congenital heart lesion. The immediate results of these operations are extremely gratifying and the future appears to be very promising.

The ductus arteriosus normally circumvents blood around the lungs while they are collapsed during fetal life. This vessel spontaneously closes within the first few months after birth, but if it should remain open an arteriovenous type of communication is thus established, which may impair the efficiency of the cardiovascular system. This abnormality causes only minor complaints during childhood or adolescence, but in adult life it often leads to serious and fatal complications, of which subacute bacterial endocarditis or cardiac failure are the commonest.

The foundations are laid in childhood for serious disease which may not become manifest until many years later. Blood rushing from the aortic arch into the pulmonary artery produces intimal thickening about the ductal orifice and in the pulmonary artery, which may later be the seat of bacterial vegetation. The vascular leak so increases the work of the left ventricle that cardiac reserve is reduced to a point where decompensation subsequently occurs. If these hazards in adult life are to be avoided, they must be warded off in childhood before permanent and irreparable damage has been done.

Working on this rationale, Gross[7] has developed a technic where the patent ductus can be exposed and surgically ligated. Studying postmortem material and practicing the operative steps on dogs, a suitable approach by way of the left pleural cavity has been found which gives an adequate exposure of the aortic arch, pulmonary artery and ductus. The feasibility of such an operation has been demonstrated by successfully exploring and ligating the ductus in 4 patients without resulting mortality. These children, aged seven, eleven, seven and seventeen years, withstood operation remarkably well and did not develop any serious complications. Following operation, all the patients showed a marked diminution in the intensity of the previously overactive heart beat. In each case the precordial thrill disappeared. The loud murmurs which were so prominent before operation were reduced to minimal ones in 2 patients, and have completely disappeared in the other 2, which were treated by placing double ligatures on the ductus. In each case the low diastolic pressure which existed before operation rose by 25 to 35 mm. of mercury to a permanently normal level. In 2 cases there was no appreciable change in the size of the heart, but in the other 2 there were diminutions in its transverse diameter of 0.5 and 1.0 cm., respectively. Calculations of the blood flow were made in 2 cases by determining oxygen concentrations of blood samples taken from the aorta, ductus and pulmonary artery before and after ligation of the ductus. These studies showed that the peripheral circulation was greatly improved after ligation of the ductus, yet the work of the heart was greatly reduced.

The success of the operative procedure depends on recognizing this condition and differentiating it from other types of congenital heart disease. Surgery has much to offer those individuals who have a patent ductus arteriosus.

SUBDURAL HEMATOMA IN INFANTS AND CHILDREN

Ingraham and Heyl[8] have in the course of the past year presented reasons for a more radical treatment of subdural hematoma in infants and children. This is of particular interest in view of the present tendency among neurosurgeons to treat this condition by extremely conservative measures. This seems to be due to the fact that adults respond very satisfactorily to conservative treatment. When infants and children are seen only occasionally in a group of patients most of whom are adults there is a natural tendency to treat all patients in the same way regardless of age. The chief reason for a different treatment in the younger group is the presence in the majority of cases of a definite clot membrane. If no membrane is present, drainage of the hematoma through burr holes is satisfactory at any age. If even a thin clot membrane is present over the surface of an infant's brain, however, it interferes with growth, and may result in cortical atrophy, because of the rapid rate of normal growth of the brain. This opinion has been verified at this clinic by the study of older children who were not treated radically or were not treated at all. In the adult brain where the size is fixed, the presence of a membrane is not nearly so vital as long as the fluid portion of the clot is removed.

It is of interest to note that subdural hematoma is a much commoner condition in infancy and childhood than is ordinarily supposed. A series of patients ranging from two months to six years in age were studied, and the authors now advocate radical treatment consisting of the removal of the hematoma and the hematoma membranes whenever their presence has been demonstrated through burr holes. This necessitates turning down bone flaps, a procedure which these patients tolerate well if they are properly handled. When so treated,

they also resume a normal rate of physical and mental development much earlier and more completely than when they are treated by tapping and burr-hole drainage alone. It is believed that removal of the clot membrane, which is so often present, is as important as removal of the hematoma. These radical measures are often indicated in the treatment of this condition in infancy and childhood, while in the adult they would not be necessary or advisable.

FLUID NEEDS IN SURGICAL PATIENTS UNDER TWELVE

The maintenance of fluid balance is of paramount importance if good results are to be obtained in the surgery of this age group. The need for the administration of an adequate amount of fluid by the parenteral route is well recognized, and is even more important in the child than in the adult. There are certain difficulties in administering the proper amount to the small patient, and for this reason it is often inadequate. It must be pointed out, however, that bad as are the results from not giving enough fluids, there is danger of embarrassing the circulatory system in infants and children if too much is given by the parenteral route.

A good working rule is to make sure that the postoperative patient under six months of age receives and retains approximately 90 cc. of fluid per pound every twenty-four hours. For example, an infant weighing ten pounds needs approximately 900 cc. of fluid every twenty-four hours. If this infant in twenty-four hours takes and retains about 300 cc. of fluid by mouth and by rectum, approximately 600 cc. must be added by the parenteral route. These figures are not absolute, but serve as a convenient and easily remembered guide. Important factors that must be considered in each case are the degree of dehydration at the time of hospitalization and the amount of daily fluid loss from vomiting, diarrhea or the use of the duodenal tube.

Saline is best administered under the skin. Normal saline is the most easily obtainable fluid and is useful, but Hartmann's solution, which is made up of sodium lactate in physiological saline, has certain advantages. The great safety factor in administering fluid under the skin is that it will not be absorbed faster than the patient can use it.

Glucose is often necessary to meet the needs of the patient. It should not be given under the skin. Glucose thus given in concentrations of 5 to 10 per cent does not result in a sufficient absorption to be of any practical value. If higher concentrations are used, there is great danger of causing a slough. Glucose may be administered by rectum but must be given slowly and in small amounts in order to be absorbed, and the solution should never be over 10 per cent in strength.

Glucose solutions are best administered intravenously. In the infant and small child, it is often difficult to get into the vein unless proper apparatus is at hand and someone experienced in the technic of its administration is available. In giving intravenous fluids, 10 cc. per pound of body weight for an infant under one year should be the maximum amount given at any one time, and even then it should be given slowly. For a patient from one to twelve years, 5 cc. per pound should be the maximum amount given at any one time. The difficulty in administering intravenous fluid in this age group creates a great temptation to give a continuous intravenous drip after the manner so useful in adults. This method should be used with the utmost caution, as it is extremely difficult to avoid giving too much fluid too rapidly. If the drip is so arranged that it is slow enough so as not to embarrass the circulation, it may cease on account of thrombosis or collapse of the small vein used.

In any case where parenteral fluids are used for more than two or three days, the serum protein and the serum sodium should be determined daily. The serum protein should not go below 5.0 or 4.5 gm. per cent. If it is allowed to do so, edema will surely follow. The serum sodium should not be allowed to go below 130 milliequivalents per liter. One must not wait for the dangerous clinical symptoms of right-sided heart failure or peripheral or pulmonary edema to appear. If so much fluid is given that edema and low serum protein result, wound healing is likely to be seriously interfered with.

The maintenance of adequate fluid balance is of paramount importance, but the danger of giving more fluid to these small children than they can utilize must always be borne in mind. As in the use of any form of drug therapy, an overdose may be more dangerous than an insufficient dose. Even though many patients at present probably receive less than an adequate daily amount of fluid, it is well that the dangers of overloading a small patient with fluid be emphasized.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 25131

PRESENTATION OF CASE

A thirty-one-year-old single Canadian was admitted complaining of swelling of the neck and palpitation.

About fourteen years before admission the patient first noticed a swelling in the right side of his neck. This did not increase in size and produced no significant symptoms. There were no choking sensations and no pain. There was no progression in the size of the swelling for ten years preceding his admission. Four years ago he entered the Out Patient Department where he was advised to have his thyroid operated on. This he refused to do. Ten weeks before entry following a heavy meal he had a warm sensation in the chest and epigastrium, developed a feeling of faintness, which lasted about six or eight minutes, and broke out into cold perspiration. He entered an outside hospital where he was told that he was all right and was advised to eat light meals and take soda. A week later he had a similar episode about one hour after a light evening meal. Physical examination in the Out Patient Department on the following day showed enlarged nodes in the neck. These were thought to be tuberculous. An electrocardiogram was normal. The blood pressure was 130 systolic, 80 diastolic. He came to the tumor clinic of this hospital three weeks before entry, at which time the nodes in the neck were again noted. The left lobe of the thyroid was small but contained a 1-cm. nodule. The right lobe was the size of a small lemon, was firm, and was distinct from the lateral mass of nodes. His weight had remained normal. He had not had chest pain, dyspnea, night sweats, cough or dysphagia.

Physical examination showed a well-developed and nourished man in no distress. There was a large matted mass of non-tender nodes occupying the anterior and posterior cervical regions on the right except for the nodule in the thyroid. The right lobe of the thyroid measured 3 cm. in diameter and was firm. The nodes measured up to 2.5 cm. in diameter and were movable beneath the skin. The left side of the neck was essentially normal. Examination of the chest and abdomen was negative. The blood pressure was 140 systolic, 100 diastolic.

The temperature was 99°F., pulse 80, respirations 24.

Examination of the urine was negative. The blood showed a red-cell count of 5,460,000 with 85 per cent hemoglobin, and a white-cell count of 11,000 with 86 per cent polymorphonuclears. A blood Hinton test was negative.

X-ray films of the chest showed displacement of the trachea to the left, apparently by a mass which lay in the right mediastinum and extended up into the right neck.

On the eighth hospital day an operation was performed.

X-RAY INTERPRETATION

DR. AUBREY O. HAMPTON: There is a definite displacement of the esophagus and trachea to the left and thickening of the soft tissues in front of the trachea. I think we ordinarily call such a picture a tumor of the thyroid.

DIFFERENTIAL DIAGNOSIS

DR. GEORGE A. MARKS: It seems to me the problem here is primarily whether to consider the mass of nodes separately or with the nodular thyroid as all part of one picture. The history is not quite clear as to just when the nodes were first found. The man is said to have had a mass in his neck for fourteen years and on examination in the Out Patient Department four years before his present entry a thyroidectomy was recommended. It was only when he returned here that any mention is made of the nodes, so I think it is fair to assume that the mass in his neck which was said to have been present fourteen years was probably the right lobe of the thyroid.

Going back to consider the nodes themselves, if they are to be considered separately, it seems to me the diagnosis lies between tuberculosis and lymphoblastoma, Hodgkin's disease or lymphosarcoma. The course since the development of the nodes might be consistent with tuberculosis although there is nothing striking in the physical examination in that regard. Tuberculous nodes are frequently tender, and episodes of tenderness may be associated with them, even though they do not go on to abscess formation. I think one of the striking aspects of the whole case is the fact that this man apparently was in such good health when he was admitted to the hospital. The course of tuberculous adenitis would probably hold episodes of fever. Hodgkin's disease, although it may be restricted to one side of the neck for a fairly long time, usually has a more debilitating course with relapses and remissions than is evidenced in the

history in this particular case. Fever is common, often to a fairly high degree. The other aspect which strikes me is the fact that he had so few symptoms referable to the mass itself, or to the nodes. The history records specifically the absence of chest pains, of difficulty in swallowing or difficulty in breathing. Hodgkin's disease in the mediastinum is very frequently accompanied by rather severe symptoms from pressure either on the esophagus and trachea or on the various branches of the sympathetic chain.

I do not believe that either of these diagnoses fulfills the picture as it is given here and the third diagnosis which we certainly ought to consider is that of a primary lesion of the thyroid. It is fair to assume that he had an enlarged thyroid for a period of years. He did not show signs of hyperthyroidism. His weight is said to have remained steady. There is no mention of the other usual signs and symptoms of hyperthyroidism. He did have elevation of blood pressure and that is about all that can be even faintly suggestive of hyperthyroidism. If it is not hyperthyroidism then the various tumors of the thyroid must be considered. The one tumor which seems to fit all the various findings is the tumor classed as malignant adenoma. This may appear at an early age. The initial enlargement of the thyroid may remain without much change in size over some period of years and it eventually metastasizes. When it does, it may go by direct extension or by lymphatic invasion to the cervical nodes or into the mediastinum or by extension in veins into the lungs or bone. The tumor and its metastases may both be very highly differentiated and may suggest normal thyroid tissue. It is not a rapidly progressing and debilitating process such as adenocarcinoma or papillary carcinoma of the thyroid.

There are one or two symptoms which I attempted to fit into the picture. These are two attacks which were described as a warm sensation in the chest and abdomen and a feeling of faintness. These occurred eight to nine weeks before he came to the hospital. I thought that possibly a tumor located in the superior mediastinum in relation to the sympathetic chain might produce temporary pressure on it due to rapid change in size caused by hemorrhage or softening. These tumors of the thyroid are prone to cystic degeneration. A hemorrhage into the cyst might explain sudden change in size. The blood picture suggests infection. I tried to explain the slight leukocytosis and the slight amount of fever also on the basis of hemorrhage into a degenerative cyst or to necrosis. X-ray films show the deviation of the trachea. They do not show calcification in the region of the neck in which the nodes are located, which one might expect with tuberculosis. The location of the mass is very high. There is no evidence of Hodgkin's disease elsewhere in the chest and the mass is above the hilar nodes and in a region which is perfectly compatible with a substernal thyroid mass. So I will rest with that diagnosis, a malignant adenoma of the thyroid with cervical and mediastinal metastases.

Dr. Paul D. White: Why could not these two attacks of faintness be paroxysmal tachycardia brought on by a full stomach plus the pressure?

Dr. Tracy B. Mallory: It could be possible. Anatomically I cannot answer that.

Dr. Francis M. Rackemann: Could the attack be due to pressure on the carotid since that is not far away?

Dr. White: I think a warm sensation is more apt to indicate a fast cardiac action than a slow one.

Dr. Richard H. Wallace: I sent him in from the tumor clinic and my original impression was that the nodes and the thyroid were distinct. I only saw him the one time in the tumor clinic. I thought that the firm matted nodes felt more like tuberculous ones than anything else, that the thyroid was distinct and that it probably was a simple colloid goiter. Dr. Richard Miller operated, I believe, and even after operation thought that it was a benign thyroid. The picture did not become clear until the slides were examined.

Clinical Diagnosis

Neurofibroma of right neck.

Dr. Marks's Diagnosis

Malignant adenoma of the thyroid.

Anatomical Diagnosis

Papillary adenocarcinoma of thyroid.

Pathological Discussion

Dr. Mallory: The specimen which was removed consisted of a lobe of thyroid and a chain of enlarged cervical lymph nodes that measured 17 cm. in length. There were about fourteen separate nodes that we counted making up the chain and also three or four separately submitted nodes which were not connected with the main chain. All the various specimens showed the same histologic picture. It was that of a papillary adenoma of the thyroid. We know that all these papillary adenomas are potentially malignant just as the rather similar looking tumors of the ovary are, although the usual histologic signs of malignancy may not be very obvious. These cases often present a difficult problem to both the surgeon and

the pathologist. We have seen several cases in which a single nodule was removed from the neck as a biopsy and in which the question comes up, Is it metastasis to a lymph node or is it tumor of accessory thyroid tissue? We know, of course, that accessory thyroid tissue is particularly prone to develop into papillary cyst adenoma. It may be very difficult to tell from looking at the sections whether one is dealing with primary tumor rising in accessory thyroid tissue or with metastasis from a primary focus in the thyroid itself. In this case with the multiple gland involvement there is of course no question. We have seen a number of these cases in this hospital and they all have run a very slow course such as this one has. I do not see how we can tell how long the primary tumor has been present and over how long a period metastases have been developing. I think one is prone to think of carcinoma of the thyroid as one of the few forms of cancer that frequently metastasizes by the blood stream, and we therefore forget the possibility that it not infrequently metastasizes via the lymphatics just as ordinary cancer does.

DR. MARKS: Was the mass in the mediastinum a lower pole of the node?

DR. MALLORY: The lower pole of the long chain of nodes.

CASE 25132

PRESENTATION OF CASE

A sixty-five-year-old, Austrian Hebrew building wrecker entered complaining of right chest pain, cough and blood-streaked sputum.

One week before entry, at night, without previous cough, sputum or pain, the patient developed "grippe" and "cold." He had recovered ten days previously from a similar condition. The last attack was soon followed by a sudden onset of sharp, stabbing pain in the right side, involving the right lumbar region, right axilla and right shoulder, associated with a cough productive of dark-red blood mixed with mucus. Coughing increased the pain. He had a temperature of 100°F. but no chill. His physician gave him a hypodermic for the pain and referred the patient to an outside hospital where he continued to raise dark-red sputum. His chest was strapped, without relief. The blood white-cell count ranged from 9000 to 13,000. The temperature ranged from 100.5 to 99°F., the pulse from 110 to 70. Respirations remained at 20. X-ray films of the chest showed density obscuring the right costophrenic angle, suggestive of fluid, and haziness of the left costophrenic angle. There was also some density of the right hilus in the region of the descending bronchus. The heart shadow was normal. An electrocardiogram showed no evidence of heart disease. A chest tap yielded some bloody fluid which showed no tumor cells.

The patient denied any weight loss, weakness or shortness of breath. He had worked considerably with iron and steel.

Physical examination on entry showed a well-developed and nourished man in no distress. Examination of the heart was negative. The blood pressure was 130 systolic, 80 diastolic. Coarse intermittent rales were heard in the right base posteriorly and flatness and dullness were made out in the inferior lateral portion. The remainder of the physical examination was noncontributory.

The temperature was 98°F., the pulse 80, the respirations 20.

The urine examination was negative. The blood showed a red-cell count of 4,400,000 with 80 per cent hemoglobin, and a white-cell count of 9400 with 72 per cent polymorphonuclears. The non-protein nitrogen of the serum was 26 mg. per cent. The sputum was bloody and non-odorous.

X-ray films of the chest showed a local area of density in the right costophrenic angle near the posterior axillary line, which was in contact with the pleura, moved with respiration and was convex toward the lung root. There was hazy density around the shadow, which extended upward along the costophrenic angle and extended into the interlobar pleura. The anterior costophrenic angle on the right side was shallow. There was a dense line extending from the area of consolidation upward and medially toward the lung root. There was no mediastinal shift and the remainder of the lung fields were clear.

On the second hospital day the patient's condition remained essentially unchanged. The following day the patient complained of substernal pain and soon thereafter shortness of breath. Ten minutes later he was pale, cyanotic and showed definite air hunger. The cervical veins were dilated. The pulse was not obtainable at the wrist and there was no measurable blood pressure. The lungs were clear. The heart rate was very slow, the sounds feeble. The patient's condition rapidly became worse and he died about forty minutes later.

X-RAY INTERPRETATION

DR. RICHARD SCHATZKI: There is a lesion in the right lower lobe posteriorly. An almost triangular area of consolidation is seen close to the pleural surface, with a linear area of increased density going toward the hilus. I came in too late

to hear the history. I should like to know how long that has been going on.

Dr. Theodore C. Pratt: It started a week before entry with cough, chest pain and hemoptysis and with bloody fluid in the pleural cavity.

Dr. Schatzki: Of course that makes the diagnosis definite.

Differential Diagnosis

Dr. Pratt: The differential diagnosis deals with those conditions that produce sudden hemoptysis with associated chest pain of pleural character. It seems to me we can rule out quickly any serious abdominal condition. No mention is made of symptoms or signs referable to the abdomen. Of the chest conditions producing sudden pain and hemoptysis several can be rapidly ruled out. There is no question of pneumonia, with or without empyema, in view of the symptoms and subsequent course. Tuberculosis can also be ruled out because of the essentially negative lung fields elsewhere and the negative past history. Pulmonary abscess or bronchiectasis can be ruled out for the same reason. He had no history of foul sputum and there is nothing in the clinical history to suggest it. A large mediastinal tumor which might erode through into the pleura or involve the lung can also be ruled out because of an apparently normal mediastinum by x-ray. An aortic aneurysm of the thoracic aorta I believe can be definitely excluded because of the completely negative examination so far as the heart and blood pressure are concerned. In addition an aneurysm in order to produce sudden death here would have to rupture into the pleura and produce sudden massive hemorrhage. His death as we see later was not due to sudden massive hemorrhage. In reading over the x-ray report and before I had seen the films I had considered the possibility of carcinoma of the lung, either of the bronchus or of the alveolar type, which we can now probably rule out on the basis of the x-ray alone. He had had no previous cough or dyspnea of long duration and no loss of weight and the fluid removed from his chest showed no tumor cells. Certainly carcinoma of the lung or of the bronchus is very unlikely.

That leaves us with only the diagnosis of pulmonary infarct secondary to a small embolus and followed later by a larger and fatal pulmonary embolus. This diagnosis seems to fit most of the data given. The only objection is that we are given nothing in the history or physical findings suggestive of deep phlebitis. There is no mention of tenderness of the legs or edema. If this were an operative case we could assume with a great deal of assurance that deep phlebitis existed either in the iliac, femoral or popliteal vein. However, in this case, we have evidence of a definite infection of some type, perhaps respiratory, of about ten days' duration which might have been the etiologic factor for a phlebitis that occurred, possibly, in the pelvic veins. We shall assume that he did have phlebitis. The only other explanation for an infarct due to embolus would be that he had a thrombosis in his right heart or a primary thrombosis in the pulmonary artery, of which there is no evidence. So a pulmonary infarct representing the first episode seems reasonable. The temperature and the elevation of the white count fit in with that, as do his physical findings. The x-ray film, which is interpreted as showing a wedge-shaped area with the apex toward the hilus, also agrees, and I imagine that the extension over to the hilus is a collection of interlobar fluid. Would that be consistent?

Dr. Schatzki: This is a line that Dr. Hampton and Dr. Castleman are studying. It is probably due to atelectasis.

Dr. Pratt: The type of pain here is consistent with a pulmonary infarct of the right lower lobe and simply means diaphragmatic pleural irritation. I cannot explain the radiation into the lumbar region although I believe that it might occur. Certainly pleural effusion is consistent and with a large infarct can very well be blood stained. I believe, therefore, that the diagnosis is pulmonary infarct secondary to a small embolus occluding one of the pulmonary artery branches and followed by a fatal pulmonary embolus almost completely occluding the pulmonary artery, both emboli arising from the same source, probably a deep phlebitis.

Dr. Champ Lyons: This patient came in with a diagnosis of cancer of the lung, but on entry the brevity of his illness led us to believe he had a pulmonary infarct. We examined his extremities very carefully at the time and were unable to localize any definite signs of peripheral phlebitis.

Clinical Diagnosis

Pulmonary embolism and infarction.

Dr. Pratt's Diagnoses

Pulmonary infarct and fatal pulmonary embolus — both secondary to a deep phlebitis.

Anatomical Diagnoses

Pulmonary embolism, massive.
Pulmonary infarction, recent, right lower lobe.
Thrombophlebitis, organizing, right popliteal vein.
Arteriosclerosis, coronary and aortic, minimal.

PATHOLOGICAL DISCUSSION

DR. TRACY B. MALLORY: The only point on which I should be tempted to criticize Dr. Pratt's discussion is where he said something about phlebitis in the pelvic veins. That is possible but in the vast majority of cases with massive pulmonary emboli the phlebitis is found in the leg veins. This man had a large thrombus in the right popliteal vein, which was completely occluded. It obviously had at one time run almost all the way up the leg although the cephalad portions were broken off, which accounted for the fatal embolus found curled up in the main pulmonary artery and its right branch, so that it completely blocked the circulation of the right lung and partially that of the left lung. The lungs showed three areas of old infarction quite separate and distinct whereas I think in the x-rays you could not be certain of more than one.

DR. PAUL D. WHITE: It is quite possible that the first attack, thought to be a cold, was the same thing.

DR. MALLORY: It is certainly possible, but it is equally possible it was a respiratory infection and that the phlebitis and all the rest were secondary to it. I do not see how we can answer that one way or the other. It is very common at autopsy to find multiple infarcts where the x-ray has been able to show only one or two of them. In the series that Dr. Castleman and Dr. Hampton have been studying recently, it is of interest that 40 per cent of all pulmonary infarcts come from cases from the medical wards that have never had a surgical operation. One has always been inclined to believe that pulmonary embolus is primarily a surgical complaint.

DR. WHITE: How many of the 40 per cent gave no clinical evidence of phlebitis?

DR. MALLORY: I should say at least 80 per cent. It is the exceptional case that shows definite evidence of phlebitis, yet in almost every case we have found a primary phlebitis in the leg veins, certainly in all the cases of massive embolism and even in most of the cases with small infarcts.

A PHYSICIAN: Recent cases from the Mayo Clinic indicate that when phlebitis makes itself known clinically the danger is essentially over.

DR. MALLORY: I should think that was very probably true. This is a type of case one sees all too often and should be a challenge to the medical profession. A patient who has had one pulmonary embolus evidenced by a demonstrable infarct is certainly in danger of subsequent ones, and perhaps a massive fatal one as in this case. It seems as though in the time interval which usually intervenes we ought to be able to advise something that would minimize the danger. As yet we have absolutely nothing to offer.

A PHYSICIAN: One could tie off the affected veins if one knew where they were.

DR. MALLORY: There is practically never any clinical evidence of thrombosis.

DR. WHITE: I have had three patients this year whose veins I have had tied off.

DR. MALLORY: What has been the result, from the point of view of circulation?

DR. WHITE: It has been good.

DR. MALLORY: They have not developed any obvious circulatory impairment?

DR. WHITE: One case is still convalescing. The others have done well.

DR. MALLORY: From the anatomical point of view it is certainly the only logical form of treatment.

DR. WYMAN RICHARDSON: From the point of view of convention one wants to think several times before putting an old person to bed without good reason. Apparently the critical period is three or four weeks. If a person is in bed for that long he is likely to stay there forever without complications.

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THE HAZARDS OF PUBLICITY

The concerted effort to reduce the pneumonia death rate more rapidly than it is reducing itself has been worthy and proper. Health authorities, eminent physicians and committees representing all sorts of interested groups are contributing to the widespread trend and desire to make what we know about this disease available to as large a part of the population as possible. Much of this effort is necessarily in the direction of popular education. Syndicated medical columns, special articles and letters in the newspapers and periodicals, broadcasts under various auspices, insurance company and commercial advertising programs, all have tried to bring a hopeful message to the people. Even if a tube of tooth paste, a cough mixture or a little eyewash has been inadvertently disposed of on the side, there has been disseminated a tremendous amount of more or less reliable information concerning our most prevalent infectious disease.

Unfortunately we have no technic by which we can simplify the truth and at the same time make it popular. To explain that there are "thirty-two kinds of pneumonia" hardly enlightens the public, nor does it state the facts. When we are cautious and precise, what we say becomes uninteresting as newspaper copy. If, on the other hand, we can devise catchwords and headlines that will attract the attention of the people we can have the publicity resources of the nation practically placed at our disposal. But acceptable headlines must be dramatic, and deliberate dramatization crushes the reality from any subject. The tendency to isolate a single passage or phrase from its context is also difficult to control; it is thus that slogans are made, and thus may be explained the fact that most of them are essentially without meaning. Such are the difficulties that beset the paths of those who would "educate" the public in the field of medicine.

Probably the most effective thrusts, for better or for worse, come about quite by accident. One such caught the editorial eyes of the country last month when a doctor said that people "just don't die" of pneumonia any more. The expression was a natural, using this noun in the American sporting sense. It was immediately hailed and used to preface the announcement of another substance claimed to have therapeutic value in pneumonia. Professional publicists could hardly have devised as apt a thought as this. It conveyed in a few easy words what pneumonia committees have conveyed only with difficulty in ponderous pages of written and spoken propaganda. It implied greater resource, greater skill and greater effort on the part of the doctor of today. It was professorial.

Effective as this pronunciamento proved to be, it still needs one minor qualification: it is not true. It cannot alter the fact that people *do* die of pneumonia, and under the best medical and nursing supervision too.

Recently a new drug, variously known as sulfapyridine, pyridine sulfanilamide and M & B 693

has been distributed for general use. Many feel that this release was premature. The drug has already received a great deal of notice as a therapeutic agent in pneumococcal pneumonia. The practicing physician is now faced with the decision whether to use serum or this new drug.

Bullowa, Plummer and Finland* have made a strong plea for caution and conservatism in accepting this new agent because of our lack of knowledge of its dangers and of the limitations of its usefulness. In view of the unprecedented publicity in the lay press accorded to the drug we are entirely in agreement with this plea. They recommend that the drug be given a thorough trial in parallel with specific serum before it is enthusiastically endorsed to the exclusion of the latter. Only a large group of observations will throw light on this problem, and it is necessary that every case possible be typed if this information is to be obtained and be of value.

Of course the incidence and mortality of the disease are decreasing, but every doctor who actually cares for pneumonia patients knows that they still die all too frequently. This should be understood by all; especially by those who have lost members of their families by pneumonia during the current year. The millennium as it concerns this disease is far, far away!

*Bullowa, J. G. M.; Plummer, N., and Finland, M.: Sulfapyridine in the treatment of pneumonia. J. A. M. A. 112:570, 1939.

THE EXPLORER PHYSICIAN

One of the greatest African travelers, who pioneered in opening up vast territories for Great Britain under the sponsorship of the African Association in the late eighteenth century, was a physician, Mungo Park. Born under humble circumstances, the seventh of thirteen children, he was apprenticed to a surgeon in Selkirk, Scotland, and received his medical degree at Edinburgh at the age of twenty. Coming to London, as most good Scots did, he fortunately fell under the eye of Sir Joseph Banks, then president of the Royal Society. Through Banks, who helped so many young men, Park obtained a post as assistant medical officer on an East Indian boat and spent some time in Sumatra. Bringing home rare plants to Banks, he was not forgotten by this leader of scientific thought in London. Two years later Banks recommended him to the African Association, and so this young man of twenty-four headed an exploration party to determine the source of the Niger River.

He was absent from England from May, 1795, until December, 1797, and the account of his travels, written in 1798, was published in 1799. The book, so modestly and simply written, became popular at once and required three editions in the same year. Handsome, six-foot Park, a little reserved in manner, became famous overnight, and one can easily imagine what receptions he must have had in London under the guidance of Banks and the members of the Royal Society.

Park, however, retired quietly to Scotland and married the daughter of his old preceptor in Selkirk, an action in every way characteristic of the man. After a few years of practice at Peebles, where he formed a warm friendship with Walter Scott, Park set out on a second expedition to the Niger. All the party were wiped out by fever or accident and Park, one of the last survivors, died in 1806. His body and records were never found.

His *Travels in the Interior Districts of Africa* (London, 1799), relating the story of his hardships during the first exploration, is a splendid narrative, written in an easy style and as fine an adventure book as one could wish for. Much of the trip was made on foot, with only a servant; he was robbed, taken prisoner, escaped, suffered from severe fever, and at last, utterly exhausted, was forced to turn back for lack of funds to purchase food. He had, however, covered three thousand miles under the most trying circumstances.

Park's high-mindedness and perseverance are now legend. His letter to Lord Camden, when he was setting out on his second and what proved to be his last effort, is often quoted: "My dear friends, Mr. Anderson and likewise Mr. Scott, are both dead; but though all Europeans who are with me should die, and though I were myself half dead, I would still persevere; and if I could not succeed in the object of my journey, I would at least die on the Niger."

MASSACHUSETTS MEDICAL SOCIETY

SHATTUCK LECTURE

Dr. Wilder Penfield, director of the Montreal Neurological Institute and professor of neurology and neurosurgery at McGill University Faculty of Medicine, Montreal, will deliver the Shattuck Lecture at the one hundred and fifty-eighth annual meeting of the Massachusetts Medical Society, to be held in Worcester on June 6, 7 and 8. His title is "Epilepsy and the Cerebral Lesions of Birth and Infancy."

SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

POSTPARTUM HEMORRHAGE

Mrs. D., a nineteen-year-old primipara, was first seen on August 17, 1938, when she was about twenty-seven weeks pregnant.

Her family history was essentially negative except for a maternal aunt who died of pulmonary tuberculosis. The patient had had the usual childhood diseases; otherwise her past history was negative. Catamenia began at thirteen, were regular with a twenty-eight-day cycle and lasted four days without pain. Her last period was February 1, 1938, making her due for delivery November 8.

Examination at the time she was first seen disclosed a well-developed and nourished young woman. Her heart was not enlarged; there were no murmurs. The lungs were clear and resonant; there were no rales. The fundus lay midway between the umbilicus and the xiphoid cartilage. The vertex was presenting and floating. The fetal heart was heard best in the right lower quadrant, the rate being 135 per minute. Urinalysis was negative except for a slight trace of albumin. The Wassermann test and urethral and cervical smears were negative.

The patient's prenatal course was normal in all respects. There was no elevation of blood pressure above 120 systolic, 85 diastolic, at any time; no edema developed, nor any other untoward symptoms. Urinalysis was normal throughout the prenatal period.

Slight uterine contractions began at 9 a. m. on November 9, and the patient was delivered of a male child weighing 7 pounds, 14 ounces, by low midforceps at 4:45 p. m. on November 10, after a labor of approximately sixteen hours and after full dilatation of slightly over three hours. A right lateral episiotomy was performed and was repaired with No. 2 chromic catgut while waiting for the placenta to separate. Fifteen minutes after the delivery of the baby a sudden profuse flow of blood occurred, apparently indicating partial separation of the placenta. The exact amount of blood was not determined, but it was estimated to be about 1000 cc. The patient went rapidly into shock, with sudden pallor, clammy skin, a weak, rapid, thready pulse and sighing respirations. Two cubic centimeters of posterior pituitary extract was immediately administered and the placenta was expressed intact by the Credé method. Two cubic centimeters of ergot was administered following the delivery of the placenta, and the fundus was gently massaged. The patient's pulse rate was 170 per minute, and her general condition was very poor. She was placed in Trendelenburg position, and 500 cc. of 5 per cent glucose in saline solution was given intravenously, and 1/6 gr. of morphine subcutaneously. When the patient was returned to bed, a clysis consisting of 1000 cc. of 5 per cent glucose in saline solution was administered. The patient's condition gradually improved, and four hours post partum her pulse had a rate of 120 per minute and was of fairly good quality. The fundus remained well contracted, and the flow was normal.

Following the initial setback the patient improved rapidly. She was given Feosol and liver extract daily and was discharged on the fourteenth day post partum, with a hemoglobin of 60 per cent and red-cell count of 3,500,000.

Comment. This case illustrates a type of postpartum hemorrhage that is frequently seen. The placenta separates quickly after the birth of the baby and is accompanied by a tremendous amount of bleeding. Fortunately oxytocics will usually control the uterus. Had this uterus not contracted well, packing might have been considered, transfusion would probably have been necessary and hysterectomy might have had to be performed. Conservative treatment is all that is necessary in many of these cases, but this case illustrates once more the need that may arise at any time for transfusion and the advisability of being prepared for such an emergency.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

LEGISLATIVE NOTES

THE CHIROPRACTORS WIN THE FIRST ROUND

Last year the Legislature set up a recess commission to study the matter, among others, of licensing chiropractors. The commission consisted of Senator Jarvis Hunt, of North Attleboro,

chairman, Representatives A. M. Bessette, of New Bedford, Oscar DeRoy, of Holyoke, and Charles Savage, of West Roxbury, and the following three men appointed by Governor Hurley: Edward W. Toomey, of Cambridge, Timothy W. Cronin, of Cambridge, and Frederick J. Hogan, of Winthrop. Hearings concerning chiropractic legislation were held in Boston, Springfield, New Bedford and Lowell. At these hearings the district legislative committees and many members of the profession tried to explain in a dignified way the importance of a single standard of education for the practice of healing. Immediately after the Legislature convened on January 4 the Attorney-General advised that members of such commissions that failed of election could no longer serve on them. As the report of the commission had not been written, due to the late commencement of the Legislature and to delay on the part of Governor Hurley in making his appointments, a new man had to be appointed to replace Mr. Savage who was not re-elected. Representative M. J. Capeles, of Pittsfield, was appointed. Mr. Capeles, who attended none of the hearings, has joined Messrs. Bessette, DeRoy and Cronin in signing a majority report in favor of the chiropractors. Messrs. Capeles and Bessette voted for the chiropractors in 1936, so that they are not new converts to the cause.

The loss of the first round in this legislative struggle is not fatal, but it means that we must go to work and work hard. The chiropractors are already flooding the legislators with letters and petitions. The bill must be stopped! We cannot do so by any half measures. Each member of the Society resident in Massachusetts should inspire at least ten letters from friends or patients to their representatives and senators opposing this legislation. As soon as the date is set for the hearing every doctor in the State should put the date on his calendar and plan either to come himself or to send a voter as a representative. The date will be put in this column as soon as it is known.

These procedures of course are extreme, and we should not have to use them. However, there are a number of legislators who are very much impressed by such interest on the part of voters and who will vote according to their estimate of their constituents' desire. Remember — it is your *duty* to be present or to be represented at the Gardner Auditorium at the State House on the day set at 10:00 a. m.

Pre-Marital Syphilis Bills

As reported to the Council on February 1 and in this column in some prior issues none of the bills for pre-marital examination for syphilis have been approved by the Committee on State and National Legislation. A practically new bill, which is deemed to be suitable, has been drawn up and agreed to by most of the parties interested in the subject. The hearing at the State House will have been held before this issue of the *Journal* is out. The committee will have put the Society on record in favor of this bill. The text follows:

House Bill 287 (revised)

An act providing for a marriage protection law, by requiring physician's examination and certificate before issuance of marriage licenses

Section 20 of Chapter 207 of the General Laws of Massachusetts is hereby amended:

Such intention of marriage shall not be accepted by the clerk or registrar until he has received from each applicant a statement signed by a registered physician, stating that said physician has examined the applicant and, if he has discovered evidence of any infectious disease which has been declared to be dangerous to the public health by the Department of Public Health, that he has informed both applicants of the nature of the disease and of the possibilities of transmitting it to his or her marital partner or to their children. Such examination by said physician shall include a standard serological test for syphilis and said test shall be made by a laboratory of the State Department of Public Health or by a laboratory approved for this test by the Department.

The physician's examination and the laboratory test shall be made not more than thirty days before the issuance of the marriage license. A marriage license issued in accordance with this act shall be valid for thirty days, after which time it shall become invalid.

Any person who shall fail to comply with the provisions of this act shall be subject to a penalty of not less than ten dollars ($10.00) nor more than one hundred dollars ($100.00).

* * *

Miscellaneous Bills

Below is listed the progress in the Legislature of some of the bills in which the Massachusetts Medical Society is interested.

Favor

S. 258. See issue of March 9. No change in status.

H. 59. Identical with S. 258.

H. 60. Bill requiring annual licensing of qualified physicians.

It was heard by the Committee on Public Health on February 9 and they have been given the ballots recently filled out by members of the Society, but no report has yet been made.

H. 72. Bill providing for the care of certain infants prematurely born. It was proposed by the Department of Public Health, and corrects defects in the previous bill.

This bill was amended to H. 2080 and has been passed to be engrossed.

H. 73. Bill providing for supplementary reporting of congenital deformities and birth injuries in infants. The bill was proposed by the Department of Public Health and

requires that supplementary reports be sent to this department.

It has been passed to be engrossed.

H. 74. Bill requiring the clerk or registrar in each city or town to give to persons who file notice of intention of marriage suitable information concerning gonorrhea and syphilis. The bill was proposed by the Department of Public Health and it contains no compulsion.

This bill was heard by the Committee on Public Health on March 28.

H. 75. Bill making various changes in the laws relating to foods and drugs. The bill was proposed by the Department of Public Health in order to bring the state law into line with the new federal act.

It will be heard by the Committee on Public Health on April 13.

H. 670. Bill providing for the issuance of certificates of approval of bacteriological laboratories by the Department of Public Health. The bill was proposed by the Massachusetts Public Health Association and is similar to the one favored by the Massachusetts Medical Society last year.

It was heard by the Committee on Public Health on March 23. There was no opposition.

H. 852. Bill requiring licensing of hospitals, convalescent homes and nursing homes. This bill was proposed by the Massachusetts Central Health Council and provides for the Department of Public Health to set up certain standards of health and enforce them.

It was heard a second time by the Committee on Public Health on March 23.

H. 1407. Bill prohibiting aliens from practicing medicine. This bill was proposed by Rep. Vaughan and is poorly written. It provides that no license be granted to an alien until his first papers have been filed but allows certain very broad exceptions.

This bill was heard by the Committee on Public Health on March 14. An amended bill has been presented.

OPPOSE

H. 287. Bill providing for a marriage protection law by requiring a physician's examination and certificate before issuance of marriage licenses. This bill was proposed by Rep. Cutler and it needs major revision before being satisfactory.

It was heard before the Committee on Public Health on March 28. (The revised bill, printed above, was favored by the Society.)

H. 551. Bill requiring that notices of intention of marriage shall be accompanied by a physician's certificate that neither party is infected with syphilis. This bill was proposed by Dr. William Frankman and also needs major revision before being satisfactory.

It was heard by the Committee on Public Health on March 28.

H. 758. Bill providing authority to the Board of Registration of Nurses to limit further training of nurses of all classes and attendants under certain conditions. The bill was proposed by Miss Josephine E. Thurlow but is against public policy.

It was heard by the Committee on Public Health on February 2 and again on March 7.

H. 759. Bill providing for training and licensing of first-class bedside nurses. This bill was proposed by Miss Josephine E. Thurlow, but is against public policy.

It was heard by the Committee on Public Health on February 2 and again on March 7.

H. 858. Bill regulating the practice of nursing. This bill was proposed by the Massachusetts State Nurses Association, and while it is better than last year's bill, some of last year's defects are still present.

It was heard by the Committee on Public Health on February 2 and again on March 7.

H. 985. Bill requiring doctors of medicine and doctors of osteopathy on the Board of Registration in Medicine. This bill was proposed by the Massachusetts Osteopathic Association and would put two osteopathic physicians on the Board.

It was heard before the Committee on Public Health on February 9. Our ballots were presented to the committee. The committee has voted leave to withdraw, and this report has been accepted in the House.

H. 986. Bill providing for a doctor of medicine and a doctor of osteopathy on the Approving Authority and the status of approvals by the American Medical Association and the American Osteopathic Association. This bill was proposed by the Massachusetts Osteopathic Association; it weakens the Approving Authority.

This bill was heard February 9 by the Committee on Public Health. Our ballots were presented to the committee. The committee has voted leave to withdraw, and this report has been accepted in the House.

H. 1401. Bill providing that certificates of vaccination or non-vaccination shall no longer be required as a prerequisite to the attendance of any child in public schools. This is a typical anti-vaccination bill.

It will be heard before the Committee on Public Health on April 4.

H. 1898. Bill providing for the establishment and administration of a system of health insurance. This bill was proposed by the State Industrial Council of the Congress of Industrial Organization (C.I.O.) and means complete state insurance medicine with a 4½ per cent pay-roll tax. It represents real regimentation of physicians.

It was heard by the Committee on State Administration on March 15. There is to be a second hearing on March 31.

CHARLES C. LUND, *Chairman,*
Committee on State and
National Legislation.

TREASURER'S REPORT COVERING REFUND DISTRIBUTION

The Treasurer of the Massachusetts Medical Society makes the following report regarding the refund to district societies for 1939.

The Council voted to distribute the sum of $4000 to district societies. The total number of payments of annual dues received by the Treasurer by March 2, to be counted for the refund, was 3916. Therefore the refund to the district societies for each paid fellow is $1.021.

The following table gives the number of payments in, and the refund to, each district as of March 23:

DISTRICT	NUMBER REPORTED PAID	REFUND
Barnstable	42	$43.07
Berkshire	105	107.30
Bristol North	50	51.22
Bristol South	155	158.33
Essex North	175	178.75
Essex South	206	210.39
Franklin	39	40.00
Hampden	236	241.02
Hampshire	57	58.37
Middlesex East	109	111.39
Middlesex North	105	107.30
Middlesex South	786	802.57
Norfolk	664	678.00
Norfolk South	113	115.44
Plymouth	117	119.53
Suffolk	527	538.13
Worcester	348	355.37
Worcester North	82	83.82
	3916	$4000.00

In 1938, for comparison, the total number of payments for the refund was 3784.

CHARLES S. BUTLER, M.D., *Treasurer.*

MEDICAL POSTGRADUATE EXTENSION COURSES

The following sessions, given by the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health, the United States Public Health Service and the Federal Children's Bureau, have been arranged for the week beginning April 3:

BARNSTABLE

Sunday, April 9, at 4:00 p. m., at the Cape Cod Hospital, Hyannis. Subject — The Indications and Contraindications for Removal of Tonsils and Adenoids. Instructor: Warren R. Sisson. Donald E. Higgins, *Chairman.*

BERKSHIRE

Thursday, April 6, at 4:30 p. m., at the House of Mercy Hospital, Pittsfield. Subject — Gonorrhea: Modern treatment of gonorrhea. Instructor: Oscar F. Cox, Jr. Melvin H. Walker, *Chairman.*

FRANKLIN

Wednesday, April 5, at 8:00 p. m., at the Franklin County Public Hospital, Greenfield. Subject — Sepsis. Instructor: A. Gordon Gauld. Halbert G. Stetson, *Chairman.*

HAMPDEN

Thursday, April 6, at 4:00 p. m., at the Academy of Medicine, Professional Building, 20 Maple Street, Springfield, and at 8:00 p. m., in the Outpatient Department of the Skinner Clinic, Holyoke Hospital, Holyoke. Subject — Bright's Disease and Hypertension: Evaluation of new therapy; diagnosis. Instructor: W. Richard Ohler. George L. Schadt, *Chairman.*

MIDDLESEX SOUTH

Tuesday, April 4, at 4:30 p. m., at the Cambridge Hospital, 330 Mt. Auburn Street, Cambridge. Subject — Whooping Cough: The present status of vaccine therapy both as prophylactic and therapeutic measure, the early diagnosis by laboratory procedures, and the treatment of complications. Instructor: Louis K. Diamond. Alexander A. Levi, *Chairman.*

SUFFOLK

Thursday, April 6, at 4:30 p. m., in John Ware Hall, Boston Medical Library, 8 Fenway, Boston. Subject — Latent syphilis: diagnosis and treatment. Instructor: C. Guy Lane. Reginald Fitz, *Chairman.*

DEATHS

BENNER — RICHARD S. BENNER, M.D., of Springfield, died March 23. He was in his sixty-fourth year.

Dr. Benner received his degree from the Harvard Medical School in 1903. He was a staff member at the Springfield and Wesson hospitals, Springfield. Among his affiliations were fellowships in the Massachusetts Medical Society and the American Medical Association and memberships in the New England Obstetrical and Gynecological Society, the American College of Surgeons and the New England Surgical Society.

His widow, two sons and two daughters survive him.

BYRNE — CLAUDIUS J. BYRNE, M.D., of 1066 Main Street, Worcester, died March 18. He was in his fifty-sixth year.

Born in Moultonboro, New Hampshire, he attended high school in Manchester, New Hampshire, and was graduated from Tilton Seminary, Tilton, New Hampshire. He received his degree from Tufts College Medical School in 1910.

Dr. Byrne was one of the two senior surgeons at the Worcester City Hospital and last January was elected president of the Worcester City Hospital Staff Association. He was also surgeon at Wickwire Spencer Steel Company. His memberships included the Massachusetts Medical Society, the American Medical Association and the American College of Surgeons.

His widow, a son and a sister survive him.

DRURY — JOHN N. DRURY, M.D., of 9 Central Street, Lowell, died November 19, 1938. He was in his fifty-eighth year.

Born in Lowell, he attended the local schools and received his degree from New York University College of Medicine in 1904. He also graduated from the Bellevue Medical School. Dr. Drury was a staff member of the Bellevue Hospital for eight years and later conducted a private practice in New York City until 1914, when he returned to Lowell.

A great deal of his time was given to the tuberculosis clinic conducted by the city health department. Dr. Drury was a fellow of the Massachusetts Medical Society and of the American Medical Association.

His widow, a daughter, a son, his father, three brothers and several nieces and nephews survive him.

GRAY — ELIZABETH T. GRAY, M.D., of 149 Warren Street, Roxbury, died March 24. She was in her seventy-seventh year.

Born in Roxbury she attended Girls' High School and was graduated from the Posse School of Physical Education in 1892. She received her degree from the Woman's Medical College of the New York Infirmary for Women and Children in 1895.

After her retirement ten years ago she was appointed consulting surgeon of the New England Hospital for Women and Children and at the time of her death was a director and faculty member of the Posse School.

A nephew survives her.

McLAUGHLIN — JOSEPH I. MCLAUGHLIN, M.D., of 92 Walnut Avenue, Roxbury, died March 26. He was in his seventy-ninth year.

Born in Boston he was educated in the Boston schools and at Boston College and received his degree from the Harvard Medical School in 1890. Dr. McLaughlin was appointed physician at the Charlestown State Prison in 1891 and had continued his private practice in Roxbury until several months ago.

He was a fellow of the Massachusetts Medical Society and the American Medical Association.

Two sisters survive him.

WOODALL — CHARLES S. WOODALL, M.D., of Brandon, Vermont, died March 26. He was in his forty-seventh year.

He received his degree from the Harvard Medical School in 1924. For twelve years he served as assistant superintendent of the Fernald State School in Waverley and resigned to become the head of the Brandon State School. He was a diplomate of the American Board of Neurology and Psychiatry and a member of the National Board of Medical Examiners.

Dr. Woodall was a fellow of the Massachusetts Medical Society and the American Medical Association and held memberships in the American Psychiatric Association and the New England Society of Psychiatry.

His widow survives him.

MISCELLANY

"YOUR HEALTH" BROADCASTS

The next series of "Your Health" broadcasts, sponsored by the American Medical Association and the National Broadcasting Company and heard over the Blue Network each Wednesday at 2:00 p. m., is entitled "Health Education." It consists of four broadcasts as follows:

April 5. Don't Believe Everything!
Fallacies and popular beliefs that are not true and that influence behavior in a manner detrimental to health.

April 12. Learning to Live.
Elements of mental hygiene; getting along with people; adjustment to environment.

April 19. "Accidents Don't Just Happen——"
Accidents in the home and on the highway and ways to avoid them.

April 26. What Is a Doctor?
The characteristics of a reputable physician as distinguished from cultists, quacks, fakers, faddists or exploiters.

MANIFESTO BY BALKAN MEDICAL UNION

The Balkan Medical Union, in session at Istanbul, for the fifth Medical Week,

HAVING TAKEN INTO CONSIDERATION the terrible sufferings which a total war will bring upon the civil population of open towns with a total lack of any adequate means of protection, and

HAVING DISCOVERED that even in its restricted form the project of "sanitary towns" has not yet been adopted, and that all efforts made to protect civilians against chemical warfare have till now remained as proposals only, and that even the protocol prohibiting the use of asphyxiating gas has not yet been ratified by all nations, therefore

HAS DECIDED to address itself to doctors of every nation with an appeal to take active measures and to fulfill this professional and humanitarian duty of awakening and stirring public opinion.

The Balkan Medical Union believes that only enlightened international opinion can make plain the imminence of the danger and the proved uselessness, even for the victor, of these terrible atrocities, and can thus lead to effective action. The immutable truth that *hate breeds only hate, and atrocity breeds vengeance,* must be impressed on everyone.

Prof. Dr. Bensis, Dr. Scaramanga (Athènes), Dr. Zika Markoviç, Prof. Dr. K. Sahoviç, Dr. M. Simoviç (Beograd), Prof. Dr. Gheorghiu, Dr. Popescu Buzeu (Bucarest), Prof. Dr. Akil Muhtar Ozden, Prof. Sedat Tavat, Prof. Dr. A. Süheyl Ünver (Istanbul).

CORRESPONDENCE

UNITED JEWISH CAMPAIGN

To the Editor: At this time of extreme need for the Jews, the question is raised, "What are Jews of America doing to help?" It has come to my attention that a nation-wide campaign is being launched by this race to raise a tremendous sum of money to help in giving relief.

On March 19, the United Jewish Campaign officially opened with the object of raising money for the purposes listed below:

1. For the relief of the dislocated groups in Europe, under the direction of the Joint Distribution Committee.

2. For the United Palestine Appeal, the funds of which are to be used in co-ordinating settlement and protecting in every way both the settled population and the refugees who have flocked to Palestine to the number of 200,000 in the past few years.

3. For the National Co-ordinating Committee Fund, which has the purpose of maintaining and distributing refugees in the United States and elsewhere.

In addition, certain re-educational and cultural programs are being financed. The scope of the relief work to be done is world wide, and the suffering to be relieved is abysmal.

Naturally, the Jews will take care of the larger part of the financing of this immense problem. However, I believe that there are many doctors in this community who may wish to show their sympathy and good-will by making a small contribution toward this campaign which is humane in the most complete meaning of the word and which is to uphold the dignity and defend the liberty of human beings.

Those who are interested to do so may send a note or a check to Dr. Abraham Myerson, Lister Building, Boston, who is chairman of the Medical Committee.

HILBERT F. DAY, M.D.

412 Beacon Street,
Boston.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

To the Editor: In addition to the articles enumerated in our letter of February 7 the following have been accepted:

Abbott Laboratories
- Abbott's Nicotinic Acid Tablets 50 mg.
- Abbott's Nicotinic Acid Tablets 100 mg.

Arzol Chemical Co.
- Mercurochrome Applicators — Arzol

Eli Lilly & Co.
- Ampules Metycaine 2 per cent, 30 cc., in rubber-stoppered vials

Mead Johnson & Co.
- Mead's Nicotinic Acid Tablets, 20 mg.

Medical Arts Laboratory
- Rabies Vaccine (Killed Virus) packages of 7 vials

Merck & Co., Inc.
- Nicotinic Acid — Merck

Wm. S. Merrell Co.
- Ephedrine Sulfate — Merrell
- Ampule Solution Ephedrine Sulfate — Merrell, ¾ gr. (0.05 gm.), 1 cc.
- Ampule Solution Mercury Succinimide — Merrell, 1/6 gr. (0.01 gm.), 1 cc.

The National Drug Co.
- Immune Globulin (Human)

The Upjohn Co.
- Solution Procaine Hydrochloride 2 per cent, 30 cc. vials
- Solution Procaine Hydrochloride ½ per cent with Epinephrine, 5 cc.
- Ampule Solution Procaine Hydrochloride 2 per cent with Epinephrine, 1 cc.
- Ampule Solution Procaine Hydrochloride 2 per cent with Epinephrine, 3 cc.
- Solution Procaine Hydrochloride 1 per cent with Epinephrine, 30 cc. vials
- Solution Procaine Hydrochloride 2 per cent with Epinephrine, 30 cc. vials

PAUL NICHOLAS LEECH, *Secretary.*

535 North Dearborn Street,
Chicago, Illinois.

NOTICES

REMOVAL

BERNARD ZUCKERMAN, M.D., announces the removal of his office to 1804 North Avenue, Bridgeport, Connecticut.

ANNOUNCEMENT

DR. WILLIAM E. BROWNE, of 587 Beacon Street, Boston, has resumed in full his former duties of practice following an illness of several months.

UNITED STATES CIVIL SERVICE EXAMINATIONS

Associate Medical Officer, $3200 a Year

Applications must be on file with the United States Civil Service Commission at Washington, District of Columbia, not later than April 10.

Applicants must have had at least one year of general internship, or one year in a special branch. They must have been graduated from a medical school of recognized (Class A) standing with the degree of M.D., subsequent to May 1, 1934.

Associate Health Education Specialist, $3200 a Year
Assistant Health Education Specialist, $2600 a Year

Applications must be on file with the United States Civil Service Commission at Washington, District of Columbia, not later than April 17.

Applicants for either position must have successfully completed a full four-year course leading to a bachelor's degree in a college or university of recognized standing.

Applicants for the position of assistant health education specialist must show at least two years of postgraduate study successfully completed toward a certificate, diploma or a degree in hygiene or public health in a college or university of recognized standing.

* * *

The necessary forms may be obtained from the Secretary, Board of the United States Civil Service Examiners, at any first-class post office, from the United States Civil Service Commission, Washington, District of Columbia, or from the United States Civil Service district office.

BOSTON DOCTORS' SYMPHONY ORCHESTRA

Rehearsals of the newly organized Boston Doctors' Symphony Orchestra, conducted by Nicolas Slonimsky, are held every Thursday evening at 7:30 at Hampton Court Hotel, 1223 Beacon Street, Brookline.

Membership is still open. All physicians, dentists and medical and dental students who are interested should communicate with Dr. Julius Loman, Pelham Hall Hotel, Brookline (BEA 2430).

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p.m. on Thursday, April 6, in the amphitheater of the Peter Bent Brigham Hospital, Dr. William P. Murphy will give a medical clinic. Practitioners and medical students are cordially invited to attend.

HOSPITAL COUNCIL

The annual meeting and luncheon of the Hospital Council of Boston will be held at the Palmer Memorial, New England Deaconess Hospital, 195 Pilgrim Road, Boston, on Tuesday, April 11, at 12:30 o'clock.

Miss Carrie M. Hall, R.N., will report on her investigation of nursing homes.

NEW YORK ACADEMY OF MEDICINE

The Twelfth Graduate Fortnight of the New York Academy of Medicine will be held from October 23 to November 3. The subject of the Fortnight will be "The Endocrine Glands and Their Disorders."

The program will include clinics and clinical demonstrations at many of the hospitals of New York City, evening addresses and appropriate exhibits. The evening sessions at the Academy will be addressed by recognized authorities in their special fields.

A complete program and registration blank may be secured by addressing Dr. Mahlon Ashford, New York Academy of Medicine, 2 East 103rd Street, New York City.

DELTA OMEGA LECTURE

The eleventh Delta Omega Lecture of the Department of Biology and Public Health of Massachusetts Institute of Technology will be held at the Massachusetts Institute of Technology, Cambridge, on Friday, March 31, at 5:00 p. m.

Dr. John E. Gordon will speak on "Public Health in the Balkans."

The lecture is open to all who are interested.

AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS

The twenty-fourth annual meeting of the American Association of Industrial Physicians and Surgeons with the American Conference on Occupational Diseases and Industrial Hygiene will be held at the Hotel Statler, Cleveland, Ohio, June 5, 6, 7 and 8. A program of timely interest and importance will be presented by speakers of outstanding experience in all the medical and engineering problems involved in industrial health. A cordial invitation is extended to all whose interests bring them in contact with these problems. Information regarding hotel accommodations, and so forth, may be obtained from A. G. Park, Convention Manager, 540 North Michigan Avenue, Chicago.

JOSEPH H. PRATT DIAGNOSTIC HOSPITAL

Bennet Street, Boston
Auditorium, 9–10 a. m.

Medical Conference Program

Tuesday, April 4 — Low-Back Pain. Dr. J. D. Adams.
Wednesday, April 5 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, April 6 — The Status of Tuberculosis in the British Isles. Dr. S. V. Pearson.
Friday, April 7 — Adrenal Function and Angina Pectoris (Theory and Therapy). Dr. William Raab.
Saturday, April 8 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, April 11 — Physical Examinations of Groups. Dr. R. W. Buck.
Wednesday, April 12 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, April 13 — Metrazol Therapy in Dementia Praecox. Dr. Arthur Berk.
Friday, April 14 — Here and There in Endocrinology. Dr. Fuller Albright.
Saturday, April 15 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, April 18 — Some Newer Aspects of the Treatment of Acidosis. Dr. Nelson R. Saphir.
Thursday, April 20 — Medical Social Service Case Presentation. District Service and Social Service Staff.
Friday, April 21 — Ascorbic Acid. Dr. Allan Butler.
Saturday, April 22 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, April 25 — Diagnosis of Atypical Jaundice. Dr. William Dameshek.
Wednesday, April 26 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, April 27 — Alcohol: Chemical tests for alcoholism. Dr. Sydney Selesnick.
Friday, April 28 — Heredity and Environment in Relation to Intelligence, Personality and Mental Disease. Dr. Abraham Myerson.
Saturday, April 29 — Hospital Case Presentation. Dr. S. J. Thannhauser.

FAULKNER HOSPITAL CLINICOPATHOLOGICAL CONFERENCE

The monthly clinicopathological conference of the Faulkner Hospital will be held on Thursday, April 6, at 5:00 p. m.

There will be a discussion of cases by Drs. J. S. Hodgson and G. M. Morrison.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, April 3

Monday, April 3

First Annual Regional Convention of the Association of Medical Students. Harvard Medical School.

*4 p. m. Physicians and medical students are cordially invited to attend a clinic presented by the medical, surgical and orthopedic services of the Infants' and Children's hospitals, in the amphitheater of the Children's Hospital.

Tuesday, April 4

*9-10 a. m. Joseph H. Pratt Diagnostic Hospital. Low-Back Pain. Dr. J. D. Adams.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

Wednesday, April 5

*9-10 a. m. Joseph H. Pratt Diagnostic Hospital. Hospital case presentation. Dr. S. J. Thannhauser.

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

Thursday, April 6

*9-10 a. m. Joseph H. Pratt Diagnostic Hospital. The Status of Tuberculosis in the British Isles. Dr. S. V. Pearson.

*3:30 p. m. Medical clinic at the Peter Bent Brigham Hospital.

5 p. m. Faulkner Hospital clinicopathological conference.

Friday, April 7

*9-10 a. m. Joseph H. Pratt Diagnostic Hospital. Adrenal Function and Angina Pectoris (Theory and Therapy). Dr. William Raab.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

12 m. Urological conference, Massachusetts General Hospital, lower outpatient amphitheater.

Saturday, April 8

*9-10 a. m. Joseph H. Pratt Diagnostic Hospital. Hospital case presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff rounds of the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

March 31 — Delta Omega Lecture. Notice above.

March 31 — Boston Dispensary, luncheon meeting of the clinical staff. Page 542, issue of March 23.

April 1, 2 and 3 — First Annual Regional Convention of the Association of Medical Students. Page 541, issue of March 23.

April 2 — Health Lecture. Quincy City Hospital. Page 363, issue of February 23.

April 4 — Lawrence Cancer Clinic. Page 541, issue of March 23.

April 4-29 — Joseph H. Pratt Diagnostic Hospital. Medical Conference Program. Notice above.

April 6 — Medical Clinic at the Peter Bent Brigham Hospital. Page 580.

April 6 — Faulkner Hospital clinicopathological conference. Notice above.

April 11 — Hospital Council. Page 580.

April 13 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

April 21 and 22 — New England Health Education Institute. Page 542, issue of March 23.

May 7-15 — International Congress of Military Medicine and Pharmacy. Page 501, issue of September 29.

May 12 and 13 — American Heart Association. Page 542, issue of March 23.

May 13-16 — American Board of Obstetrics and Gynecology. Page 457, issue of March 9.

May 14-20 — American Physicians' Art Association. Page 404, issue of March 2.

May 15-19 — American Medical Association. St. Louis, Missouri.

May 22, 23, and 24 — American Association for the Study of Goiter. Page 405, issue of March 2.

June 5, 6, 7 and 8 — American Association of Industrial Physicians and Surgeons. Page 581.

June 6, 7, and 8 — Massachusetts Medical Society. Worcester.

June 12-17 — Symposium on the Public-Health Significance of the Virus and Rickettsial Diseases. Page 125, issue of January 19.

June 26-29 — National Tuberculosis Association. Page 936, issue of December 8.

September — Boston Psychoanalytic Institute. Page 450, issue of September 22.

September 11-15 — American Congress on Obstetrics and Gynecology. Page 938, issue of December 8.

September 15-28 — Pan-Pacific Surgical Association. Page 863, issue of November 24.

October 23 - November 3 — New York Academy of Medicine. Page 581.

Fall, 1939 — Temperature Symposium. Page 218, issue of February 2.

District Medical Societies

ESSEX SOUTH

April 5 — Addison Gilbert Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Ethan Allan Brown. Subject: Allergy.

May 10 — Annual meeting. Salem Country Club, Peabody.

NORFOLK DISTRICT

March 28 — Page 493, issue of March 16.

SUFFOLK

March 29 — Joint meeting with New England Pediatric Society. Page 541.

April 26 — Annual meeting in conjunction with Boston Medical Library, at 8:15 p. m. Election of officers. Program and speakers to be announced.

WORCESTER

April 12 — Page 542, issue of March 23.

May 10 — Worcester Country Club — annual meeting.

BOOK REVIEWS

The Open Mind: Elmer Ernest Southard, 1876-1920. Frederick P. Gay. 324 pp. Chicago: Normandie House, 1938. $5.00.

Why a biography of Elmer Ernest Southard? To his friends, associates and students, this question seems entirely unnecessary. The biographer, Dr. Frederick P. Gay, professor of bacteriology at Columbia University College of Physicians and Surgeons, certainly never had any doubt in his own mind that the life and work of Southard made him a proper subject for a biography. Not because Southard was the first Bullard Professor of Neuropathology at Harvard Medical School and the first director of the Boston Psychopathic Hospital; not merely because he was an outstanding chess player and the favored disciple of William James and Josiah Royce; but because, in the words of the biographer: "Ernest Southard was a great deal more than an intellectual prodigy who scattered inspiration and, it must be confessed, at times dismay, among the orthodox. *He was a great human being.* He was a unique, directive and often a predominating influence in the lives of many men and women. He seldom left anyone, who passed more than a fleeting moment in his company, indifferent."

From Latin School days, Dr. Gay believed that his friend was unique. He kept every letter from Southard, and he finally concluded that Southard was a "genius." In essence, the writing of the biography was a work of love and probably there was a compulsive drive toward its accomplishment. The biographer, being a scholar and scientist, a friend from boyhood days, a co-student and colleague, and for a time a co-worker, and throughout Southard's life, a confidant, is eminently equipped not only to present the life and character of Southard, his views and his philosophy, but also to interpret his scientific accomplishments and to evaluate his work and his contribution to medicine and to society. This is what Dr. Gay has tried to accomplish.

The book is divided into seventeen chapters dealing with the subjects of inheritance, early life, college years, medical training, personality and career. Two chapters are devoted to Southard's work as a pathologist and neuropathologist. Individual chapters deal with Southard as a state officer, as the director of the Boston Psychopathic Hospital, in psychiatric social work, as an etymologist, philologist, philosopher and psychologist and as an educator and psychiatrist.

In the course of the narration, Dr. Gay sketches much of the history of psychiatry in its broadest aspects during the important period 1910-1920, and all who have an interest in this subject will find much of value. In the minds of those who worked in this field during that period, the challenge of Southard's view will again be brought to life. To those of the newer generation much background material will be uncovered.

How well has Dr. Gay wrought? This is a hard question for the reviewer — an old disciple of a revered chief — to answer. If Dr. Gay succeeds in impressing the reader with the extraordinary influence that Southard had on his students and associates, he will have accomplished a great deal. It is obvious that no biographer can seem completely adequate to one who lived close to Southard and was under his influence for years. But unquestionably it is a good biography. It is pleasant reading; it is anecdotal; it is valuable as a history of psychiatric thought; and it is a thoughtful portrayal of an important and interesting personality.

Teachable Moments: A new approach to health. Jay B. Nash. 243 pp. New York: A. S. Barnes & Co., 1938. $1.50.

From the publisher's blurb one might assume that this is an epoch-making treatise on the subject of health training. It is not. Dr. Nash is not a physician; he is a popular teacher of physical education, and aiming at popularity, is rather given to oversimplification and the employment of catchwords and phrases. Yet his do's and don't's of health, though in no sense new, are sound on the whole, and he has not permitted himself to become the propagandist of this or that fad or cult. His book may be recommended with safety if not with enthusiasm.

The New International Clinics. Original contributions: clinics; and evaluated reviews of current advances in the medical arts. Edited by George M. Piersol. Vol. 4, N. S. 1. 350 pp. Philadelphia, Montreal, New York: J. B. Lippincott Co., 1938. $3.00.

The fourth volume of the *New International Clinics* upholds the standard set by previous contributions. There are a number of original contributions on a wide variety of topics. In addition, three reports from clinics are given and one review. This review is perhaps the most important article in the whole volume, being a complete summary of our knowledge of the so-called Cushing's syndrome, with an extensive bibliography. In addition, there are good articles on the placenta; polyneuritis; vitamin B_1 in the American diet; hypertension; and the length of life of cardiac cases. The volume, as usual, is well illustrated by both pictures and diagrams, and the bibliographies are carefully chosen.